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THE PHYSIOLOGY AND CLINICAL SIGNIFICANCE OF VENOUS PRESSURE.

By ANTHONY PROUST, M.B., B.S., M.R.C.P. (London), M.R.C.P. (Edinburgh),

Honorary Physician, Canberra Community Hospital, Canberra.

THE physiological principles underlying circulatory failure are constantly under review. The instruments used to measure cardiac functions and to assess circulatory states are becoming more complex, and are rarely available to the clinician at the patient's bedside. Hence the problems of early diagnosis, accurate prognosis and progress in therapy of cardiac failure are being relinquished to the physiologist and cardiologist, who, by their research methods, are isolating themselves from the clinician and, more importantly, from the patient in his own environment. Those of us who have the opportunity to study the course of heart disease during its often slow evolution must turn this to advantage. Arnott (1959) wrote: "I think cardiology is the very last specialty which should be sharply circumscribed in its dimension and served by jealous exclusive devotees."

In the present investigation, the primary assessment of the patient's circulatory state rested upon his symptoms and physical signs, which, in the great majority of cases, have been repeatedly observed over three or more years. In most cases, electrocardiograms were taken and X-ray studies of the heart were performed. The venous pressure was measured directly in the cubital fossa by one observer using the same equipment. In all, 466 readings were taken from 226 patients, of whom 115 were in heart failure. Sir Thomas Lewis (1946) wrote:

> Direct measurement of the venous pressure will often provide an invaluable guide to a patient's state [of systemic congestion]; repeated readings will often indicate clearly the course of the malady.

In this investigation, the venous pressure measurements will be discussed in relation to the patients' progress during the years from 1955 to 1960.

Historical Survey.

It is agreed that Stephen Hales in 1733 made the first direct measurement of venous pressure by inserting a glass tube into the jugular vein of a horse and observing the height to which the blood rose (Eyster, 1926; Deitchman and Canter, 1952).

It was not until 1906 that von Recklinghausen estimated the venous pressure in man. He placed an air-tight chamber over a superficial vein on the back of the hand, sealed it to the skin, raised the pressure within the chamber and, as the vein collapsed, observed the pressure on an aneroid manometer.

In 1910 Moritz and Tabora in Germany were the first to measure the venous pressure directly in man by inserting a needle into a peripheral vein, and measuring the level of saline in a manometer which was in free communication with the lumen of the vein.

In the United States, Winsor and Burch (1946), Hussey and Jeghers (1947) and Hitzig (1945) have done a great deal of research into the value of venous pressure measurements. The equipment has been simplified and the technique standardized, so that over the past 20 years, series of measurements by different investigators can be accurately compared. However, the need for further investigation has been underlined by Landis and Hortenstine, who wrote in 1950:

> Consideration of the multiple interlocking factors which affect and are affected by venous pressure reveals how limited is our quantitative information concerning this measurement.

Functional Anatomy.

Veins are thin-walled tubes which carry the blood from the tissues to the heart. The walls of the veins, like those of the arteries, are in three layers: (i) a lining intima; (ii) a media of smooth muscle and connective tissue; (iii) an outer adventitia, mainly of connective tissue. Again like arteries, the veins are supplied by the sympathetic nervous system. Veins differ from arteries in being thin-walled, collapsible and distensible and, in the extremities, by having numerous valves within their lumina. The cross-sectional area of the veins is more than twice that of the arteries (Franklin, 1928).

Normal Physiology of Venous Flow and Blood Pressure.

The most important factor controlling blood flow is the forward output of the heart. During ordinary everyday activity, the ventricles are called upon frequently and suddenly to vary their output according to exercise and changes in the emotions and the metabolism of the body. They do this normally by increasing their rate of contraction and their stroke volume in response to the increased venous return, according to Starling's law.

Equally important is the circulating blood volume, which in health is constant within narrow limits. It has been estimated that 70% of the circulating blood is in the venous system, 18% in the arteries and 12% in the capillaries (Landis and Hortenstine, 1950). However, at any instant volumetric shifts may occur between the various vascular segments, and blood may be mobilized from the storage pools in the splanchnic area. If the demands for increased circulation are sustained, not only do the heart rate and the stroke volume rise, but the circulating blood volume increases. The distensible venous system can accommodate this extra blood and the venous pressure rises only slightly. However, if the circulatory demands are prolonged and excessive, then the venous pressure may rise significantly and itself become a factor in increasing cardiac output and maintaining circulatory equilibrium. This elevation of the venous pressure is physiological, because it is a stimulus to a healthy ventricle to increase its output under temporary conditions of excessive volume load.

Richards (1947) summarizes this well:

> Starling's law applies to the human circulation as well as one could expect a physiological principle developed from the performance of a denervated heart-lung preparation; the more the heart is exposed and reacts to nervous, vasomotor and metabolic stimuli, the less it behaves in accordance with the law; the more the heart fails to respond to such influences and becomes simply a pressure-volume muscle preparation, the more closely it follows the law.

The role of peripheral resistance is important in venous blood flow and pressure. With lowered resistance in the arteriolar and capillary beds, more blood enters the veins, which dilate to accommodate it. The venous return increases and the cardiac output rises. The venous pressure remains stable.

In 1928 Franklin established that veins were in a state of tone—venomotor tone—and that this was a factor altering vascular volume, blood flow and blood pressure throughout the venous system. This has been confirmed by other workers, who have selectively influenced venomotor tone with amyl nitrite and "Priscol", causing changes in venous pressure (Weiss *et alii*, 1937; Braun and Fryd, 1951; Rado, Gonda and Kovacs, 1958).

The negative intrathoracic pressure acting upon the thin-walled veins of the upper part of the abdomen and the thorax increases the rate of venous blood flow towards the heart. When the negative intrathoracic pressure is added to the positive venous pressure in the great thoracic veins, the result is a reduced peripheral venous pressure.

Finally, gravity, venous valves and the massaging effect of skeletal muscles all influence venous blood flow and pressure, but as they are all eliminated, as far as possible, by the technique of measurement used in this study, they will not be further discussed. Interstitial tissue pressure is mentioned briefly, because in old and emaciated patients the lack of this pressure may possibly reduce venous pressure. However, generally in health or disease it would be of no importance.

The blood pressure and the rate of flow through the vascular circuit have been estimated as follows (Best and Taylor, 1943; Bainbridge and Menzies, 1940): in the large arteries the pressure is 1200 to 1350 mm. of water, and the flow 500 mm. per second; in the small arteries the pressure is 1100 to 1200 mm. of water, and the flow 50 mm. per second; in the arterioles the pressure is 270 to 1100 mm. of water, and the flow 5 mm. per second; in the capillaries the pressure is 125 to 270 mm. of water, and the flow 0·5 mm. per second; in the veins pressure is 5 to 125 mm. of water, and the flow 10 mm. per second.

The greatest fall in pressure occurs in the arteriole, which separates the high-pressure arterial segment from the low-pressure venous bed. The arterial segment is essentially a supply system with rigid walls, into which blood is injected from the left ventricle at high velocity and under high pressure to meet a formidable resistance towards the periphery. The venous system is a drainage bed and reservoir, both distensible and contractile, in which a much greater volume of blood under low pressure flows at slow speed to the right ventricle. One may therefore expect the left ventricle to be most efficient under pressure loads, and the right ventricle to work maximally under volume loads (Sinclair-Smith, 1958).

Venous Pressure in Cardiac Failure.

Cardiac failure may be defined as the inability of the heart to supply the body with sufficient blood for its metabolic needs. Eyster describes it as "that condition in which venous pressure exceeds the range within which the heart is capable of responding, by increased work, to increased venous load".

Functionally, the types of cardiac failure may be classified as follows (from Huckabee, Casten and Harrison, 1950):

> 1. Disorders of cardiac filling due to (*a*) excessive venous return, e.g. hyperkinetic circulatory states, (*b*) mechanical hindrance to ventricular filling, e.g. mitral stenosis.
>
> 2. Disorders of cardiac emptying due to (*a*) increased resistance to forward output, e.g. hypertension, (*b*) failure of myocardial function due to degenerative changes, e.g. ischemic heart disease.

Cardiac failure may be acute or chronic and may involve the left or the right ventricle or both. The predisposing disease may be in the heart (the myocardium, the endocardium or the pericardium), in the vascular circuit, in the blood, in the lungs, in the kidneys or in the endocrine glands, but in the final analysis, cardiac failure is a breakdown of the left or the right ventricle in its function as a pump.

There are four major and well-established features in cardiac failure: (i) inadequate forward cardiac output (Altschule, 1938; Schroeder, 1950); (ii) impaired renal

excretion of water and salt (Futcher and Schroeder, 1942; Merrill, 1946); (iii) increased circulating blood volume (Warren and Stead, 1944; Gunton and Paul, 1955); (iv) increased venous pressure (Gibson and Evans, 1937; Harrison, 1938; Warren and Stead, 1944). Not all these features necessarily occur in all patients with cardiac failure—some appear early, some later, and often the change may be simply a relative one. A patient may die in heart failure with a diminishing forward cardiac output which is still within the range of normal (McMichael, 1952).

The measurement of cardiac output is a research procedure which at present has no clinical application in the diagnosis or management of cardiac failure.

The impairment of renal excretion of water and salt may be due to the reduction of the renal blood flow and glomerular filtration rate by a diminished forward cardiac output (Merrill, 1946; Liddle *et alii*, 1955). It may also arise from tubular dysfunction resulting from hormonal changes or raised renal venous pressure (Blake *et alii*, 1949).

It appears that retention of water and retention of salt do not necessarily go hand in hand—in some cases there is primarily water retention, and in others primarily retention of sodium. The posterior pituitary antidiuretic hormone and the adreno-cortical hormone aldosterone probably play an important role. However, at present the clinical application of renal physiology in cardiac failure is limited to measurements of the body weight in relation to the urine output, of serum electrolyte levels and of blood urea levels.

Circulating blood volume is usually increased in cardiac failure as a direct result of water retention, and its measurement would be most helpful clinically. However, this is, like the measurement of the forward cardiac output, a research procedure.

The final major physiological feature of cardiac failure is venous hypertension, which can be measured accurately at the bedside with simple equipment and without danger or discomfort to the patient.

Less well-established physiological features of cardiac failure are the changes in peripheral vascular resistance and venomotor tone. Increased peripheral vascular resistance has recently been demonstrated (Wood *et alii*, 1955). This would increase the pressure load on the left ventricle. Increased venomotor tone has also been recently suggested (Braun and Fryd, 1951; Halmagyi *et alii*, 1952). This would decrease the area of the venous bed and increase venous blood flow, thus increasing the volume load on the right ventricle.

Clinically, heart failure is usually discussed in terms of the ventricle most affected. The isolated failure of the left ventricle may be quite sudden in onset, as in acute myocardial infarction, occurring in a few score beats. The forward output of the left ventricle falls while that of the right ventricle continues unchanged, causing a pooling of blood within the pulmonary vascular circuit. The pulmonary capillary pressure rises and some unknown factor (possibly neurogenic) increases the capillary permeability (Hayward, 1955). The patient develops a short, dry cough and a wheeze, and proceeds rapidly to a state of severe orthopnœa. The skin is pale, cyanosed and moist, the pulse is rapid, the blood pressure is normal or elevated and pulsus alternans appears. Gallop rhythm at the apex of the heart and scattered lung crepitations are usually heard. The diminished forward output of the left ventricle may return to normal quickly and permanently, leaving behind no persistent circulatory changes. The systemic venous pressure is probably normal in most cases.

However, the failure of the left ventricle may be associated with pronounced circulatory changes throughout the whole vascular circuit. This occurs when the left ventricle has been working under increased pressure loads for long periods, as it does in cases of hypertension and of aortic valvular disease. The left ventricle adapts itself to this by hypertrophy and dilatation, and compensatory congestive changes may appear in the systemic veins while both ventricles remain competent. Hence, from a pressure load on the left ventricle, there develops a volume load on both ventricles. The patient's exercise tolerance diminishes, but cardiac competence may still be preserved precariously for some considerable time. However, sudden effort, emotion, infection or arrhythmia causes a fall in the forward output of the left ventricle, while the right ventricle continues to respond to the increased volume and pressure loads in the systemic circuit. A major shift in blood volume to the pulmonary circuit occurs and the patient is in danger of death from pulmonary œdema. In these cases, the venous pressure should be raised not only during, but before, the attack of acute pulmonary œdema. The prognosis in these cases is poor — repeated attacks of left ventricular failure occur, and finally congestive cardiac failure supervenes.

Failure of the right ventricle is usually associated with, or preceded by, some degree of failure of the left ventricle, as has been described above. Only in cor pulmonale and in some forms of congenital heart disease does it occur in the absence of failure of the left ventricle. Right ventricular failure is usually slow in onset, because it is a failure under volume and pressure loads. These loads are slow in building up because of the complex physiological factors involved, and the right ventricle has a means of adaptation to them at almost every level.

The pressure load—the less important—is transmitted through the pulmonary vascular tree by the failure of the left ventricle. The volume load may have started to build up months, and even years, before congestive cardiac failure is first diagnosed. This volume load is at first compensatory—not in the physiological sense, as in sustained exercise in a healthy person, but in the functional sense of helping a failing heart to remain competent. This increased volume load stems firstly from changes in venomotor tone. However, when the failing left ventricle reduces the renal arterial blood flow and the glomerular filtration below a critical level, salt and water are retained in excessive quantities. The circulating blood volume and systemic venous pressure then rise beyond the level at which they act as a stimulus to cardiac output. At this point, the increase in renal venous pressure may impair tubular reabsorption of water and salt and a vicious cycle is created, increasing the volume load on a failing right ventricle. When the venous pressure rises above 200 mm. of water, significant tricuspid incompetence occurs. Any further increase in venous pressure causes an actual fall in the forward output of the right ventricle. When this occurs, the distribution of circulating blood is overwhelmingly on the venous side of the circulation, and all the hallmarks of congestive cardiac failure are readily apparent — engorgement of the neck veins, hepatomegaly, ascites and œdema of the dependent parts. At the same time, the patient is dyspnœic in proportion to the degree of left ventricular failure.

Attention has recently been drawn to the so-called hyponatræmic heart failure (Schroeder, 1949; Mickerson and Swade, 1959). The clinical picture is familiar—emaciation, œdema, cyanosis, orthopnœa, engorgement of the neck veins, ascites, jaundice and hæmoptysis. The urine is scanty, and heavy albuminuria is present. The administration of digitalis and diuretics provokes little response; both ventricles appear to be in irreparable failure. Some success has recently followed steroid therapy and new interest has been shown in this condition. The serum sodium level is low, the blood urea level is raised and the retention of water in the body is grossly increased. That this is not necessarily the final stage of a long drawn-out congestive cardiac failure, or the end-result of the unrelenting use of mercurial diuretics, is known to clinicians who have watched the condition appear in an unpredictable fashion. The mechanism of this type of cardiac failure is possibly a metabolic disturbance of the intracellular osmolarity, with a consequent increase in the extracellular water. More water than salt is retained in the body, so that while the total amount of salt in the body remains high,

the serum sodium level falls. Whether this metabolic disturbance is due to pituitary and adrenal factors, or is essentially renal in origin, has not yet been established.

There is a final clinical subdivision of cardiac failure recognized by Stead (1951) as congestive failure "with œdema due to an abnormal distribution of the fluid normally present in the body". The cardiac output is low, while the circulating blood volume and venous pressures are normal. The following explanations may be advanced: (i) The right ventricle may fail, and the right auricular pressure may rise, with little or no elevation in the peripheral venous pressure (Richards and Cournand, 1942). (ii) High peripheral vascular resistance reduces the venous blood pressure and flow (J. E. Wood, 1955). (iii) A fall in venomotor tone may occur, which allows pooling of blood in the venous circuit (Sharpey-Shafer, 1944). (iv) Abnormal extravascular fluid distribution may occur, as a result of changes in cellular metabolism (Elkinton and Squires, 1951). Probably old age, low metabolic requirements and poor tissue tone all play a part. The clinical picture is of œdema of the dependent parts, often quite marked, and an adequate exercise tolerance over short distances. The most common symptoms are tiredness, dizziness and swelling of the ankles.

Hence in summary, there are five recognizable types of heart failure. Firstly, failure of the left ventricle may be subdivided into two types: (i) isolated failure of the left ventricle as a pump under a pressure load (the venous pressure is normal in this type); and (ii) failure of the left ventricle with systemic congestion—that is, failure under both pressure and volume loads—and an elevated venous pressure. Secondly, failure of the right ventricle may be subdivided into two types: (i) chronic right ventricular failure under increased volume and pressure loads (the venous pressure is elevated in this type); and (ii) failure of the right ventricle as a pump under pressure loads only. Thirdly, there may be failure of both ventricles associated with hyponatræmia. The venous pressure may be raised in this type.

An attempt has been made to correlate these theories with the clinical study and venous pressure measurements of 115 patients in cardiac failure.

Technique of Direct Venous Pressure Measurements.

The venous pressure is measured in an ante-cubital vein. The patient is allowed to rest for 15 minutes in a comfortable supine position, the head supported by a thin pillow and the right arm abducted at an angle of 45° from the body and resting on an arm-board. Care is taken that the selected vein is at the level of the junction of (i) a vertical plane passing through the fourth costochrondal cartilage with the patient in the supine position, and (ii) a horizontal plane running through the midpoint of the antero-posterior diameter of the chest. Any tight clothing is loosened or removed, and the patient is encouraged to relax, being assured that the actual procedure is painless and short in duration.

The following apparatus has been assembled and sterilized: (i) a 10 ml. syringe and a gauge 18 needle; (ii) a three-way stopcock to which the needle and syringe are attached; (iii) a glass manometer calibrated to 320 mm. with an extension to 600 mm.; (iv) a 10 ml. vial of 2·8% sodium citrate solution. The syringe is filled with the sodium citrate solution and the needle is inserted into the selected ante-cubital vein. When blood has been aspirated freely into the syringe, the manometer is attached upright to the stopcock, which is then turned so that the manometer is filled with the citrate solution from the syringe. The stopcock is turned again, to place the citrate in the manometer in direct communication with the lumen of the vein. The patient is encouraged to relax and breathe normally. The level of the citrate solution falls, and when it reaches a stable level, with small fluctuations caused by respiration, this is recorded as the direct venous pressure in millimetres of water.

In some cases, the following manœuvre is performed. After the direct venous pressure is read, an assistant applies firm pressure with both hands to the right upper quadrant of the patient's abdomen for a period of one minute. The fluid level is read again and recorded as a separate measurement (Pasteur-Rondot manœuvre).

The Present Series.

Four hundred and sixty-six measurements of venous pressure have been performed on 226 patients during the period from 1955 to 1960. This figure includes 83 measurements which were taken after the Pasteur-Rondot manœuvre had been carried out. In the series were 122 males and 104 females, whose ages ranged from nine to 93 years and averaged 59 years.

The patients have been divided into the following groups: (i) those without demonstrable heart disease (control group—51 patients); (ii) those with demonstrable heart disease (*a*) without symptoms or signs of heart failure (75 patients), (*b*) in left ventricular failure (61 patients), and (*c*) in right ventricular failure (54 patients). Fifteen patients appear in more than one group because their circulatory status changed during the long period of observation.

The ætiology of the heart disease was as follows: (i) ischæmic, degenerative and hypertensive heart disease (108 cases or 61·7%); (ii) rheumatic heart disease (41 cases or 23·4%); (iii) cor pulmonale (10 cases or 5·7%); (iv) hyperkinetic circulatory states (8 cases or 4·6%); (v) primary rhythm changes (4 cases or 2·3%); and (vi) congenital heart disease (4 cases or 2·3%).

The incidence of the various types of heart disease in this series is similar, in the major classifications, to that reported by White (1953) in the United States and to that quoted by Wood (1955) in the United Kingdom. Hypertensive, ischæmic and degenerative heart disease accounted for 61·5% of the clinical cases of heart disease reported by Wood and for 64·8% of those of White. Cases of rheumatic heart disease comprised 20% of Wood's series and 23·5% of White's series. There was wide divergence between all three series in the incidence of the less common heart diseases.

The Control Group.

The average venous pressure for the control group of 51 patients was 94 mm. of water. The standard deviation was 3·67. Five readings were above 125 mm. of water; two of these patients were thyrotoxic (venous pressures 135 mm. and 146 mm. of water) and two were apparently healthy (venous pressures 136 mm. and 126 mm. of water). One patient was anæmic, having a hæmoglobin level of 9·8 grammes per 100 ml. (venous pressure 130 mm. of water).

Twenty-two of these patients were healthy and symptomless. The average venous pressure for this group was 88 mm. of water.

Twenty-nine of the patients were considered to have healthy hearts, even though they complained of breathlessness (24 patients) or ankle œdema (five patients). After full investigations had been carried out, the final diagnoses were established as follows: chronic bronchitis, asthma and emphysema (10 cases); anxiety states (six cases), obesity, anæmia and thyrotoxicosis (three cases in each category), chronic nephritis (two cases), Banti's syndrome (one case) and toxæmia of pregnancy (one case). The average venous pressue in this group was 97 mm. of water.

Heart Disease Without Heart Failure.

Seventy-five patients suffered from heart disease without heart failure. The decision to exclude heart failure from the diagnosis was sometimes difficult, but it was always made without reference to the venous pressure. It was based upon the patient's observed ability to continue his occupation without overt symptoms or signs of heart failure.

The average venous pressure in this group was 109 mm. of water; the standard deviation was 3·74. Twenty-seven patients had venous pressures above 125 mm. of water.

The ætiology of the heart disease in these 75 cases was as follows: (i) ischæmic, hypertensive and degenerative heart disease (38 cases); (ii) rheumatic heart disease (31 cases); (iii) primary rhythm changes (four cases); (iv) congenital heart disease (two cases).

One might have expected, over an average observation period per patient of nearly two years, that a significant number of the 27 patients with raised venous pressures would go into heart failure. So far this has happened in only three cases.

CASE 97.—A shop assistant, aged 48 years, first presented in 1955 with a cerebral embolus associated with paroxysmal auricular fibrillation and mitral and aortic rheumatic valvular disease. In June, 1958, a routine reading of his venous pressure was 152 mm. of water; he was asymptomatic, with a good exercise tolerance. In February, 1959 he had two sudden, severe attacks of cardiac asthma (venous pressure 156 mm. of water), but he has remained well while being treated with digoxin, quinidine and diuretics. His venous pressure was 154 mm. of water in January, 1960. He was last examined in January, 1961.

CASE 129.—A trainee nurse, aged 19 years, was examined in 1955, one year after a mitral valvotomy had been performed. A routine venous pressure measurement taken in August, 1958, was 133 mm. of water; she was asymptomatic. In January, 1960, she developed acute pulmonary œdema when two months pregnant (venous pressure 209 mm. of water). After treatment, her venous pressure fell to 135 mm. of water and her confinement was normal.

The following three patients have been followed up for over two years after the venous pressure was first found to be elevated.

CASE 29.—A woman, aged 44 years, the mother of two children, works full-time in an office, doing all her own housework at night and at week-ends. She has rheumatic heart disease with mitral stenosis. Her venous pressure was 172 mm. of water in August, 1958. She was given digoxin on five days each week and has continued with this treatment because of a lessening of her fatigue. She was last examined in January, 1961.

CASE 103.—A mining engineer, aged 36 years, has rheumatic heart disease with aortic incompetence and mitral stenosis. He has occasional attacks of auricular fibrillation. In May, 1958, his venous pressure was 170 mm. of water. He was free of symptoms. In addition to his carrying on his job, he was fencing a grazing property in rugged country at week-ends. He is being treated with digoxin and oral diuretics, and his venous pressure is unchanged (February, 1959). He was examined in January, 1961, when he denied the presence of any symptoms.

CASE 218.—A station hand, aged 20 years, had acute rheumatic carditis in 1955. He developed aortic incompetence and mitral stenosis. His venous pressure in October, 1958, was 163 mm. of water. He continues to do sheepwork, cattle-branding, fencing and rabbiting without complaint.

Eleven of these 75 patients have developed cardiac failure while under observation; of these, only three were in the group of 27 patients with venous pressures above 125 mm. of water, and all developed left ventricular failure. On the other hand, eight of the 48 patients with venous pressures below 125 mm. of water have developed failure, four each left ventricular failure and congestive cardiac failure.

Nine of the 75 patients have died. The causes of death were acute coronary occlusion and cerebral thrombosis (each accounting for three patients), pulmonary embolism, left ventricular failure and congestive cardiac failure (each accounting for one patient).

Left Ventricular Failure.

Venous pressure measurements were taken from 61 patients in left ventricular failure. The average reading was 138 mm. of water; the standard deviation was 4·75 mm. Thirty-eight patients had venous pressures above 125 mm. of water.

The ætiology of the left ventricular failure was as follows: (i) ischæmic, degenerative and hypertensive heart disease (48 cases); (ii) rheumatic heart disease (10 cases); (iii) hyperkinetic circulatory states (three cases).

Seventeen patients suffered acute coronary occlusions before developing left ventricular failure. Their venous pressures averaged 158 mm. of water. Thirteen of this group survived and five have returned to their original occupations.

Thirteen patients with hypertensive heart disease and left ventricular failure had average venous pressures of 160 mm. of water.

Eighteen patients with ischæmic heart disease without infarction or significant hypertension had average venous pressures of 117 mm. of water while in left ventricular failure.

Measurements of venous pressure have been taken as a routine measure from patients with acute myocardial infarction. In this way, cardiac failure is less often overlooked and repeated measurements ensure that the treatment is adequate.

CASE 1.—A grazier, aged 71 years, had an acute myocardial infarction in June, 1955. Twenty-four days later, after repeaed myocardial infarctions, he developed acute pulmonary œdema. His venous pressure rose to 183 mm. of water. After treatment with venesection, digitalis, diuretics and anticoagulants he recovered, and his venous pressure fell to 92 mm. of water after his discharge from hospital. On two occasions since his discharge, he has had milder episodes of left ventricular failure, which have responded to treatment. He still supervises his grazing property, riding horseback, and his venous pressure was 68 mm. of water in May, 1958. He remains on anticoagulant treatment (February, 1961).

CASE 38.—A company director, aged 78 years, with diabetes mellitus, had an acute myocardial infarction in February, 1957. In September, 1957, he developed left ventricular failure and his venous pressure rose to 194 mm. of water. His condition responded to treatment, and his venous pressure fell to 138 mm. of water. He remained active until his second coronary occlusion in August, 1959, when he died suddenly during the convalescent stage.

CASE 114.—A carpenter, aged 44 years, was seen in a state of acute pulmonary œdema after a myocardial infarction in September, 1957. His venous pressure was 180 mm. of water. His condition responded well to treatment with venesection, digitalis, diuretics and anticoagulants, and he resumed his trade in December, 1957. In May, 1958, his venous pressure had fallen to 82 mm. of water. For two years he has been working at full pressure as a carpenter on large building projects in Canberra. He remains on anticoagulant therapy (February, 1961).

Ten patients were examined with left ventricular failure and rheumatic heart disease. The average venous pressure for this group was 139 mm. of water. Three of these patients had mitral incompetence (venous pressures 110 mm., 121 mm. and 124 mm. of water respectively) and three had undergone valvotomy operations (venous pressures 134 mm., 160 mm. and 201 mm. of water respectively). Three patients had aortic incompetence (venous pressures 92 mm., 102 mm. and 152 mm. of water respectively) and one had aortic and mitral incompetence (venous pressure 156 mm. of water).

CASE 64.—A woman, aged 23 years, underwent a mitral valvotomy operation in April, 1958, which produced a good functional result. She became pregnant in September, 1958, and remained well under close supervision, but in her thirty-second week she developed acute pulmonary œdema with a massive hæmoptysis and black cyanosis. Her venous pressure was 160 mm. of water soon after her admission to hospital. Her condition improved only slightly with treatment by venesection, digoxin, diuretics and oxygen. A Cæsarean section was performed under local anæsthesia with the patient sitting up. The cyanosis grew worse and was completely unrelieved by an oxygen tent. The patient died eight hours after operation.

Of the entire group of 61 patients in left ventricular failure, the venous pressure was below 125 mm. of water in 23 cases and above 125 mm. of water in 38. In the group of 23 patients with normal venous pressure there were five deaths, and none of the survivors has had recurrent attacks of left ventricular failure or has developed congestive cardiac failure. Among the 38 patients with raised venous pressures there were 12 deaths; eight of the survivors have had recurrent attacks of left ventricular failure and six have developed congestive cardiac failure.

There were 17 deaths in this group of 61 patient. observed for an average period of 18 months. The causes

were left ventricular failure (seven cases), congestive cardiac failure (one case), cerebral vascular accidents (four cases), acute coronary occlusion (three cases), carcinoma of the stomach and post-operative shock (one case each).

Right Ventricular Failure.

Venous pressures were measured in 54 cases of right ventricular failure. The average venous pressure was 183 mm. of water, the standard deviation being 13·6. Forty-four patients had venous pressures above 125 mm. of water.

The ætiology of the underlying heart disease was as follows: (i) ischæmic, degenerative and hypertensive heart disease (32 cases—average venous pressure 190 mm. of water); (ii) cor pulmonale (10 cases—average venous pressure 120 mm. of water); (iii) rheumatic heart disease (five cases—average venous pressure 248 mm. of water); (v) hyperkinetic circulatory states (five cases—average venous pressure 216 mm. of water); and (vi) congenital heart disease (two cases).

Seventeen patients had venous pressures above 200 mm. of water. All of these patients were breathless on slight effort, although 10 were ambulant when first examined. All had œdema of the legs; in 13 cases this extended to the knees and in seven cases to the hip and abdominal wall. All the patients except one had gross hepatomegaly, and two in addition had splenomegaly. Nine had appreciable ascites. Only six were observed to have auricular fibrillation.

Thirteen of these patients are still alive after an average observation period of 27 months. Their venous pressures averaged 270 mm. of water before treatment began. Eleven are now ambulant and 10 are without gross signs or symptoms of congestive cardiac failure. Their venous pressures now average 155 mm. of water.

CASE 61.—A woman, aged 30 years, complained of swollen legs and thighs, a distended abdomen and breathlessness. She had a rapid, bounding pulse, a small goitre, slight exophthalmos and a fine tremor. Her venous pressure was 395 mm. of water. Her basal metabolic rate was +63%. A diagnosis of hyperthyroid heart disease and congestive cardiac failure was made. At rest in bed in hospital and after the injection of diuretics she had a diuresis of 143 oz. of urine in 24 hours. Six days after her admission to hospital, her venous pressure was 153 mm. of water, and 14 days later it was 104 mm. of water.

CASE 42.—A housewife, aged 40 years, has rheumatic heart disease with mitral incompetence and auricular fibrillation. When she was first examined in 1955, her venous pressure was 86 mm. of water and her weight was 166 lb. Congestive cardiac failure with ankle œdema, ascites and hepato-splenomegaly supervened in June, 1957 (venous pressure 218 mm. of water) and the patient has been treated with injectible and oral diuretics and digoxin ever since. Despite this, her venous pressure rose steadily, reaching 240 mm. of water by August, 1957, 264 mm. of water by February, 1958, 272 mm. of water by August, 1958 and 300 mm. of water by November, 1958. Chlorothiazide was then given in doses of 1 to 2 grammes daily on five days each week, with potassium chloride daily. Her body weight and venous pressure were recorded at each visit. These measurements were as follows: on November 19, 1958, 156 lb. and 300 mm. of water; on March 9, 1959, 150 lb. and 276 mm. of water; on November 20, 1959, 135 lb. and 245 mm. of water; on January 12, 1960, 137 lb. and 262 mm. of water; on January 26, 1960, 144 lb. and 258 mm. of water; on February 14, 1960, 137 lb. and 164 mm. of water; and on August 9, 1960, 131 lb. and 266 mm. of water.

The reason for the sudden marked drop in venous pressure between January 26 and February 14, 1960, was eight days' bed rest in hospital, which was considered necessary because of a sudden increase in body weight associated with hyponatræmia. The serum electrolyte levels and the blood urea levels were estimated at regular intervals and are recorded in Table I.

The emergence of hyponatræmia and hypokalæmia in this patient is probably due to sustained diuretic therapy and especially to chlorothiazide, which has been used over the past two years. However, despite the hyponatræmia, the patient's body weight and venous pressure have fallen and she remains ambulant, doing light housework and enjoying life (January, 1961).

CASE 130.—A forestry worker, aged 64 years, was examined in 1955, when an electrocardiogram revealed ischæmic heart disease. In July, 1959, he suddenly developed recurrent attacks of cardiac asthma, progressive œdema of the legs, hepatomegaly and ascites. The heart was grossly enlarged and the heart rate was 140 per minute. Auricular fibrillation was present. The urine output was low and albuminuria was present. Over the next six weeks, his condition deteriorated despite hospital rest, diuretics and digitalization. His venous pressure was 210 mm. of water, and he was found to have hyponatræmia and an elevated blood urea level. Digoxin was administered intravenously for six days, treatment with diuretics was suspended and moderate amounts of salt were allowed in his diet. Cortisone acetate—150 mg. daily—was given orally, and the dosage was reduced by 25 mg. daily after three days. The patient's improvement was rapid; his nausea disappeared, his appetite increased and his morale improved, and the pulse rate, judged by the apex beat, slowed to 70 per minute. Oral and injectible diuretics were reintroduced into his treatment after six days, with dramatic results, and cortisone therapy was suspended after 15 days. His progress is summarized in Table II.

TABLE I.

Serum Electrolytes and Blood Urea Levels during Treatment (Case 42).

Date.	Serum Sodium Content. (mEq/l.)	Serum Chloride Content. (mEq/l.)	Serum Potassium Content. (mEq/l.)	Blood Urea Content. (mg. per 100 ml.)
27/1/59 ..	140	105	3·9	—
26/5/59 ..	141	103	3·3	35
6/1/60 ..	131	98·3	3·1	37
9/8/60 ..	135	91	3·2	44

CASE 111.—A grazier, aged 59 years, suffering from hypertensive and ischæmic heart disease and left ventricular failure, was first examined in February, 1957. In May, 1958, he had an attack of acute pulmonary œdema (venous pressure 246 mm. of water). In July, 1958, the pulmonary œdema recurred and the patient remained bedridden, orthopnœic and cyanosed for nearly 12 months. The heart was grossly enlarged, the lung bases were congested and hepato-splenomegaly with ascites was present. Œdema extended to the rib margins. The venous pressure fell to 102 mm. of water (August, 1958) despite the progressive congestive cardiac failure. In February, 1959, hyponatræmia was discovered and steroid therapy was begun. After six days of steroid therapy (30 mg. of prednisolone daily in divided doses), the patient had a series of enormous diureses and lost 30 lb. in weight. His appetite improved and he became ambulant in July, 1959. Maintained on an oral dosage of digoxin, oral and parenteral administration of diuretics and small doses of prednisolone, he was able to supervise shearing on his property in October, 1959, and in 1960. His arterial and venous pressures both rose and his serum electrolyte levels returned to normal. These values are given in Table III.

CASE 199.—A housewife, aged 59 years, complained of swollen legs, breathlessness, a swollen abdomen and tiredness. Hypertensive heart disease with congestive cardiac failure was diagnosed and she was admitted to hospital on July 21, 1958, with a venous pressure of 334 mm. of water. In the first 24 hours after the administration of diuretics, she passed 239 oz. of urine. On July 29, her venous pressure was 131 mm. of water. She remains well at home, doing all the housework at a station homestead (December, 1960).

CASE 226.—A public servant, aged 60 years, developed intractable cardiac failure after myocardial infarction. Conventional therapy was ineffective, and when examined on October 31, 1960, he was cyanosed and orthopnœic, with œdema to the hips, ascites, hepatomegaly and jaundice. He was mildly diabetic, the condition controlled by diet. When admitted to hospital, he was found to be hyponatræmic. He was given insulin, prednisolone and potassium chloride, and was maintained on anticoagulants. Then digoxin and diuretics were reintroduced into his therapy. He responded well, and his urine output rose, his orthopnœa disappeared and his œdema subsided. Insulin was replaced by "Tolbutamide", given orally. His progress is summarized in Table IV.

TABLE II.

Clinical and Laboratory Findings during Treatment (Case 130).

Date.	Clinical Condition.	Venous Pressure. (mm. of Water.)	Serum Sodium Content. (mEq/l.)	Serum Potassium Content. (mEq/l.)	Serum Chloride Content. (mEq/l.)	Blood Urea Content. (mg. per 100 ml.)
6/ 9/59	Recurrent cardiac asthma; œdema to rib margins; cortisone commenced.	210	125	5·0	98·2	52
20/ 9/59	No cardiac asthma; œdema to middle of legs; cortisone suspended.	182	126	4·4	—	47
6/11/59	No œdema; ambulant; hospital discharge.	58	—	—	—	—
9/ 2/60	No œdema; good exercise tolerance.	116	126	4·1	—	44
29/ 4/60	Well.	—	141	4·1	104	44
29/11/60	Well; plays bowls.	116	—	—	—	—

The patient was discharged from hospital and allowed to go home, where, shortly afterwards, he died suddenly in heat-wave conditions. An autopsy revealed a massive fresh myocardial infarction.

Twenty-seven patients had venous pressures between 125 and 200 mm. of water. Twelve of these patients had gross pitting œdema of the legs, in seven cases extending up the thighs. Ten patients had œdema limited to the lower third of the legs, and five had minimal œdema of the ankles. Three patients had hepatomegaly. Five patients have died, all in congestive cardiac failure.

CASE 75.—A married woman, aged 36 years, had an attack of acute pulmonary congestion on July 18, 1959. She had the mitral facies, the small irregular pulse of auricular fibrillation, engorged neck veins, an enlarged pulsatile liver, an apex beat in the mid-axilla and apical systolic and diastolic murmurs. Rheumatic mitral and tricuspid valvular disease with congestive cardiac failure was diagnosed (venous pressure 170 mm. of water). The patient responded well to bed rest and to the administration of digoxin and diuretics, and eight days later her venous pressure was only 99 mm. of water. At home she was ambulant on regular maintenance therapy when she died suddenly one night in bed. At autopsy, aortic stenosis, mitral stenosis and triscuspid disease were demonstrated, together with congestion of the lungs, the liver and the kidneys.

In 10 patients, the venous pressure was below 125 mm. of water. Five of these patients had ischæmic heart disease and five had cor pulmonale. Nine had œdema of the legs, in four cases extending to the knees. None had hepatomegaly or ascites. The chief complaints were ankle œdema, tiredness and breathlessness on exertion. Three patients have died.

CASE 133.—A grazier, aged 72 years, with ischæmic heart disease and obesity, was examined in August, 1957 (venous pressure 74 mm. of water). In December, 1959, he presented with gross œdema of the legs and thighs and auricular fibrillation. His venous pressure was only 90 mm. of water. Despite the œdema, he was still working on his property. Over a period of eight weeks, while he was being treated with oral and injectible diuretics and digoxin and while he was still ambulant, his œdema gradually decreased and he felt more energetic. He returned on a routine visit in December, 1960, with extensive œdema and a weight gain of 11 lb. His exercise tolerance remained fair. His venous pressure was 160 mm. of water.

CASE 43.—A widow, aged 72 years, was under treatment for a peptic ulcer when she was weighed on January 6, 1960. Her weight was 154 lb. On January 16, she returned, complaining of breathlessness and swelling of the legs of less than one week's duration. Her weight was 155 lb. and her venous pressure 85 mm. of water. Treated with digoxin and diuretics, she lost her breathlessness and œdema by February 24, when she weighed 151 lb. and her venous pressure had risen to 99 mm. of water.

Mortality.

The 226 patients have been observed for an average of 29 months. Of the 51 patients in the control group, five have died. Of the 75 patients with heart disease who were not in failure, nine have died. Therefore, of the 126 patients who were not in failure, 14 have died, or 11·1%. Sixty-one patients had left ventricular failure, and of these, 20 died. Fifty-four had right ventricular failure, and of these, 12 died. Therefore, of the 115 patients in failure, 32 died, or 27·8%.

Pasteur-Rondot Manœuvre.

In 1885 W. A. Pasteur described the change in the jugular veins which occurs after pressure has been applied over the liver (hepato-jugular reflex). He believed this was a sign of triscuspid insufficiency. In 1898 Rondot recognized the association of this sign with right ventricular insufficiency (Deitchman and Canter, 1952). A diagnostic procedure has been evolved applying this principle to venous pressure measurements.

TABLE III.

Laboratory Findings during Treatment (Case 111).

Date.	Serum Sodium Content. (mEq/l.)	Serum Potassium Content. (mEq/l.)	Serum Chloride Content. (mEq/l.)	Blood Urea Content. (mg. per 100 ml.)	Venous Pressure. (mm. of Water.)
21/5/58	—	—	—	—	246
24/8/58	—	—	—	—	102
13/2/59	124	3·7	99	72	—
6/5/59	141	4·0	102	86	—
29/5/59	136	4·1	101	—	—
27/11/59	142	3·1	103	—	151

The Pasteur-Rondot manœuvre has been performed on 83 occasions. The results are shown in Table V.

In all 12 patients in the control group, the venous pressure fell after the Pasteur-Rondot manœuvre.

CASE 198.—A research officer, aged 41 years, was convinced he had heart disease despite several examinations by competent physicians. Repeated electrocardiograms and skiagrams were normal. His venous pressure was 126 mm. of water. After compression of the liver, the venous pressure fell to 108 mm. of water.

CASE 221.—A woman, aged 38 years, complained of ankle œdema. Her hæmoglobin value was 9·8 grammes per 100 ml. Her venous pressure was 130 mm. of water. After liver compression, the venous pressure fell to 70 mm. of water.

In the 32 patients with heart disease who were clinically not in failure, the venous pressure fell in nearly every case after compression was applied to the liver.

CASE 218.—This patient is described above in more detail. He has aortic and mitral valvular disease. In October, 1958, his venous pressure was 163 mm. of water, and after compression of the liver, fell to 158 mm. of water.

In three cases of rheumatic heart disease, the Pasteur-Rondot manœuvre provided evidence of tricuspid insufficiency in the absence of clinical signs of cardiac failure.

CASE 209.—A woman, aged 21 years, was investigated because of a heart murmur. Rheumatic heart disease with mitral stenosis was diagnosed. She had no symptoms or signs of cardiac failure. Her venous pressure was 116 mm. of water, and after liver compression, rose to 134 mm. of water.

In the 16 cases of left ventricular failure, the average movement in venous pressure was +3 mm. of water after liver compression.

In the 23 cases of congestive cardiac failure, the venous pressure rose in 22. The average movement for the whole group was +18 mm. of water. The single exception has

TABLE IV.

Clinical and Laboratory Findings during Treatment (Case 226).

Date.	Clinical Findings.	Body Weight. (lb.)	Venous Pressure. (mm. of Water.)	Serum Sodium Content. (mEq/l.)	Serum Potassium Content. (mEq/l.)	Blood Urea Content. (mg. per 100 ml.)	Weekly Urine Output. (oz.)
16/11/60	Cyanosis; orthopnœa; œdem ; ascites; jaundice.	144	300	128	3·7	65	Not available
23/11/60	Unchanged; on steroid th rapy.	156	—	135	5·2	84	217
30/11/60	Less orthopnœa.	140	—	141	4·7	—	400
6/12/60	No orthopnœa.	133	256	135	3·5	45	518
13/12/60	No ascites.	127	204	138	4·2	60	420
20/12/60	No hepatomegaly.	123	184	141	4·5	48	595
27/12/60	No œdema; ambulant.	114	158	—	—	—	540

already been described above as a case of congestive cardiac failure and normal venous pressure.

CASE 43.—When this patient was first examined (in congestive cardiac failure with gross œdema of the legs) the venous pressure was 85 mm. of water. After liver compression, the venous pressure fell to 62 mm. of water.

Arrhythmias and Venous Pressure.

Twenty-two of the 175 patients with heart disease were found to have auricular fibrillation when the venous pressure was measured. The venous pressures of these patients are shown in Table VI.

TABLE V.

Venous Pressures Before and After Pasteur-Rondot Manœuvre.

Classification of Cases.	Number of Observations.	Venous Pressure. (mm. of Water.)	Venous Pressure after Performance of Pasteur-Rondot Manœuvre. (mm. of Water.)	Variation. (mm. of Water.)
Control (51 cases) ..	12	103	89	−14
Heart disease without failure (75 cases) ..	32	118	110	−8
Left heart failure (61 cases)	16	122	125	+3
Right heart failure (54 cases)	23	159	177	+18

A patient with ischæmic heart disease was followed, with venous pressure measurements, from normal rhythm to auricular fibrillation and finally into failure.

CASE 119.—A man, aged 66 years, was examined in April, 1955, with paroxysmal auricular tachycardia. His venous pressure, with the heart in normal rhythm, was 76 mm. of water. In July, 1957, he was having long attacks of auricular fibrillation (venous pressure 133 mm. of water). In October, 1957, the fibrillation was permanent (venous pressure 194 mm. of water) and soon afterwards he had his first attack of cardiac asthma. In August, 1959, he was in congestive cardiac failure (venous pressure 207 mm. of water).

In the following case, the onset of auricular fibrillation appears to have precipitated congestive cardiac failure.

CASE 108.—An operator of earth-moving machinery, aged 47 years, collapsed fighting a bushfire at Hornsby in 1955. An electrocardiogram was read as normal. In 1956, diabetes was diagnosed and controled by diet alone. His weight fell from 225 to 210 lb. On December 30, 1959, he came for a routine check-up and apical gallop rhythm was noted. His weight was 211 lb. An electrocardiogram showed ischæmic heart disease and normal sinus rhythm. He denied the presence of any symptoms and continued working. He returned on January 13, 1960, complaining of palpitations. Auricular fibrillation was present. His weight remained constant at 211 lb. and he continued working, but was given digoxin. He returned on January 20, breathless and cyanosed, with œdema to the knees and ascites. His venous pressure was 300 mm. of water. His weight had increased to 225 lb. in seven days. He responded well to rest and the administration of diuretics and digoxin.

On January 22 he felt better and weighed only 208 lb. The venous pressure had fallen to 242 mm. of water. By January 28 the œdema had disappeared and the patient was much better, with a venous pressure of 198 mm. of water and a body weight of 202 lb. On February 1 he weighed 205 lb. and the venous pressure had risen to 209 mm. of water, but still no œdema was present. On February 6 the venous pressure had risen to 224 mm. of water and his weight to 209 lb., but he was still well. On February 12 auricular fibrillation was present, the pulse rate (judged from the apex beat) being 110 per minute.

TABLE VI.

Venous Pressures in Cases of Auricular Fibrillation.

Classification of Cases.	Number of Cases of Auricular Fibrillation.	Average Venous Pressure in Cases of Auricular Fibrillation. (mm. of Water.)	Average Venous Pressure in Cases of Normal Rhythm. (mm. of Water.)
Heart disease without failure	8	102	110
Left-sided heart failure ..	5	150	137
Right-sided heart failure ..	9	196	182

On this occasion his weight had fallen to 190 lb., although the venous pressure remained elevated, at 226 mm. of water. His healthy weight in 1954 was 196 lb.

Auricular fibrillation precipitated the cardiac failure in this patient and is a reason for the high venous pressure despite the fall in body weight and clinical improvement.

The following case illustrates the effect of paroxysmal tachycardia on a healthy heart.

CASE 210.—An hotel worker, aged 20 years, was admitted to hospital with a two-hour history of palpitation and precordial pain. An electrocardiogram showed paroxysmal auricular tachycardia with a variable block and an approximate ventricular rate of 170 per minute. The venous pressure was 34 mm. of water. The patient was given digoxin intravenously and amylobarbitone orally, and in 15 minutes he was in regular sinus rhythm. His venous pressure was 103 mm. of water two days after his admission to hospital.

In this patient with a healthy heart, the tachycardia increased the forward output of the heart to such a degree that the venous pressure fell below normal.

Repeated Measurements of Venous Pressure.

In 15 patients with competent hearts, the venous pressure measurements were repeated at intervals of at least one month. The average initial reading was 117 mm. of water and the second reading averaged 118 mm. of water.

Sixteen patients were followed, by means of venous pressure measurements, as they entered cardiac failure. In all cases, the venous pressure rose from an average reading before failure of 95 mm. of water to an average of 163 mm. of water after failure.

Forty patients were first examined in cardiac failure when the average venous pressure was 199 mm. of water.

On their improvement after treatment, the venous pressure fell in 39 cases to an average of 114 mm. of water. The single exception was a patient with congestive cardiac failure and normal venous pressure readings before and after successful treatment. (See Case 43 above.)

Discussion.

The control group in this series comprised healthy people and patients complaining of breathlessness and œdema from non-cardiac causes. There was no significant difference between the venous pressures of these subdivisions of the control group or between this control group and other series of venous pressure measurements in healthy young students (Gibson and Evans, 1937; Oschsner *et alii*, 1951). There were five cases of mild venous hypertension in the control group. In three instances it was due to hyperkinetic circulatory states (thyrotoxicosis and anæmia), and in two cases it was unexplained. When the Pasteur-Rondot manœuvre was applied in two of these cases, the venous pressure fell to normal. The occasional finding of idiopathic venous hypertension has been noted previously (Gibson and Evans, 1937). This is not surprising if one pictures the venous system as an active independent regulator of normal and pathological circulatory states.

More than one-third of patients with heart disease who clinically were in circulatory equilibrium had venous hypertension. On prolonged observation of this group, it was concluded that in some cases at least the venous hypertension was a compensating factor which served to maintain circulatory equilibrium. None of the 13 untreated patients with venous hypertension developed cardiac failure. However, this complication occurred in three of the 14 patients whose venous hypertension was treated with digoxin and diuretics. Eight of the 48 patients with normal venous pressure developed heart failure. These observations disposed of the theory that venous hypertension in this group indicated impending heart failure. When venous hypertension was found in patients with rheumatic heart disease, the diagnosis of tricuspid incompetence was considered. It was, in several instances, the first indication of this important diagnosis. However, observations on this group of patients serve to confirm that venous hypertension and tricuspid incompetence are compatible with years of circulatory equilibrium. Conversely, a normal venous pressure in a similar group of cases did not necessarily carry a good prognosis. The only previous investigation of venous pressure in patients with heart disease who were not in cardiac failure was made by Paul Wood (1936). He found that the average venous pressure in 51 patients was 70 mm. of water compared with his control group of 25 patients whose venous pressure averaged 57·5 mm. of water.

Venous hypertension was found in nearly two-thirds of the 61 patients suffering from left ventricular failure. This corresponds closely to the findings of Ernstene and Blumgart (1930), who found elevated venous pressures in 14 of their 22 patients. However, Weiss and Robb (1933) and Paul Wood (1936), in their series of 82 and 23 cases respectively, both reported venous hypertension in only a small minority. In the present series, the venous pressure was raised more often in left ventricular failure associated with myocardial infarction or hypertension than in other forms of ischæmic heart disease or in rheumatic heart disease. In myocardial infarction and hypertensive heart disease, the myocardial damage is extensive and permanent and some of the pressure load on the failing left ventricle is transferred to the right side. In other forms of ischæmic heart disease and in rheumatic heart disease, left ventricular failure is often sudden in onset and of short duration. A precipitating cause, such as arrhythmia, or infection, an emotional upset or a physical effort, is often present and can be quickly remedied. In these cases, the failure is more often confined to the left ventricle and the venous pressure remains normal. When venous hypertension is found in left ventricular failure resulting from rheumatic heart disease, tricuspid incompetence is almost certainly present. When venous hypertension accompanied left ventricular failure in the present series, only three out of 10 patients escaped recurrent attacks, the onset of right ventricular failure or death. During the same period of observation, when the venous pressure was normal in patients with left ventricular failure, then in eight cases out of 10 the patient made a sustained recovery. Apparently, venous hypertension in left ventricular failure is ineffective in maintaining circulatory equilibrium; it is a measure of the grave fall in the cardiac reserve.

In right ventricular failure, the venous pressure was raised in 44 of the 54 cases. There was a close correlation between the height of the venous blood pressure and the appearance of œdema, hepatomegaly and ascites. The occurrence of right ventricular failure with normal venous pressure was noted in 10 cases. One patient (Case 33) presented on the first occasion with right ventricular failure, œdema of the legs and a normal venous pressure of 90 mm. of water. During the subsequent 12 months, his symptoms gradually subsided, only to reappear accompanied by a sudden increase in body weight and venous hypertension of 160 mm. of water. It is difficult to assume that his cardiac failure changed from a "low-output" to a "high-output" type. It is more probable that his earlier circulatory failure represented an abnormal distribution of his normal body fluid, while on the subsequent occasion, his total body fluid was increased and abnormally distributed, and presumably associated with an increased venomotor tone. In another patient (Case 111) the reverse occurred—the patient progressed from left ventricular failure with venous hypertension (venous pressure 246 mm. of water) to failure of both ventricles with hyponatræmia (venous pressure 102 mm. of water), and finally, after reversal of the hyponatræmia, his venous pressure rose to 151 mm. of water. In this case, the changes in venous pressure did not reflect the clinical condition—the patient was in apparently irreversible circulatory failure when his venous pressure was normal at 102 mm. of water. Changes in cardiac output, circulating blood volume, total body fluid, renal function, peripheral resistance and venomotor tone all combined to produce this apparent contradiction. However, in general the venous pressure was raised in right ventricular failure to a degree compatible with the clinical signs of systemic congestion. As clinical improvement occurred, the venous pressure fell.

The Pasteur-Rondot manœuvre was confirmed as an excellent measure of right ventricular competence. Hitzig (1945) reported that if compression over the liver caused a rise of 10 mm. of water or more in venous pressure, the right ventricle was incompetent. When the right ventricle was competent, then liver compression was followed by a fall in venous pressure in 85% of 670 cases, and by no change in the remaining 15%.

In the present series, in three cases of rheumatic heart disease, in which clinically the heart was competent and the venous pressure was normal, liver compression caused a rise in venous pressure. This was interpreted as evidence of functional tricuspid incompetence. In one case of right ventricular failure, the venous pressure was normal and liver compression resulted in a further fall in venous pressure. A more recent observation in a similar case indicated that if liver compression was maintained for three minutes then the venous pressure began to rise. The tricuspid valve is sufficiently competent in such cases, so that any sudden increase in venous return results in a rise in right atrial pressure without an immediate rise in venous pressure.

Summary.

1. Four hundred and sixty-six measurements of venous pressure were made in 226 patients during the period from 1955 to 1960.

2. The average venous pressure in a control group of 51 patients was 94 mm. of water. The normal range of venous pressure was between 50 and 125 mm. of water.

3. Venous hypertension was found in nearly one-third of the patients with heart disease who, clinically, were in circulatory equilibrium.

4. Nearly two-thirds of the patients in left ventricular failure were found to have venous hypertension.

5. More that four-fifths of the patients in right ventricular failure were found to have venous hypertension.

6. The Pasteur-Rondot manœuvre of liver compression is an effective measure of right ventricular competence.

7. Venous pressure measurements, by virtue of their accuracy and simplicity, constitute a helpful aid in the diagnosis, treatment and prognosis of the diseased and failing heart.

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REMINISCENCES OF MEDICAL BRISBANE, EARLY TWENTIETH CENTURY.[1]

By A. STANLEY ROE, M.B., Ch.B. (Lond.), F.R.A.C.S.,
Brisbane.

WHEN I was invited to read the Jackson Lecture for 1960, it was suggested that the subject matter should be historical, and if possible, of medical interest. The point at which events cease to be current and begin to be historical was hard to decide, and I hope I shall fulfill the requirements of this lecture if I refer mainly to medical matters during the years in Brisbane when Dr. Sandford Jackson flourished, namely, those years from 1900 to the World War in 1914.

In 1904 I began my study of medicine at Oxford; I took a degree in physiology in 1908, then went to Guy's Hospital to do my clinical and post-graduate work and returned to Brisbane in 1913, reasonably equipped with modern medical ideas. I was then assistant to Dr. Sandford Jackson from July, 1913, to July, 1914. I must confess that when I returned to Brisbane after quite a long post-graduate spell at Guy's Hospital (ending up as president of the resident staff), I was probably somewhat pleased with myself and my professional knowledge. However, I was speedily brought down to earth in the quietest way by my father, who, as you probably know, had been a schoolmaster. My mother and father at that time lived out of town on the river up Moggill way, and one evening we returned home to find mother sick. I examined her and found she had pneumonia and pleurisy. I told Dad and said she ought to go to hospital. His only answer was: "We'll wait till the doctor comes!" The doctor came and we brought mother down to St. Helen's by boat, as the best means of transport was easily by water.

I think I should say at this stage that this lecture makes no attempt to be scientific, but is a collection of personal memories, which, I realize as I look back, are probably only now giving me a picture of what was going on medically in those early days—the days when Dr. Jackson was at his best.

In 1900 there were about 25 doctors in the metropolitan area; in 1913 there were 98 doctors in the metropolitan area; in 1960 the number stands at 850.

From 1900 onwards, thanks to enormous advances in physiology and pathology, great changes in medicine and surgery were rapidly taking place, and were not easy to keep up with; this was especially so for the practitioner who had no contact with a medical school; this was the position of the members of the profession in Brisbane at that time.

One of the senior Brisbane doctors, Dr. A. J. Turner, later made a trip abroad, and on his return began a lecture to his colleagues with the words: "We have been living in a medical backwater." Dr. Turner, I might say, was an extremely well-read student, and possessed a very inquiring mind. He was also uncompromisingly honest, as the following story will exemplify. He drove one of the early motor-cars, a wonderful model with a box-shaped, coupé type of body — very reminiscent of the wooden structures that grace so many of Brisbane's backyards. One day he happened to drive through a police trap, travelling at a snappy 26 or 27 miles per hour. He was booked and appeared in court, and was fined. The magistrate was afterwards upbraided by a friend for having fined such a man as Dr. Turner. He plaintively defended himself by saying he had done his best to let Dr. Turner off, but every time he suggested to the doctor that he was going to an urgent case, Dr. Turner replied: "Oh no, I was just going home." So justice had to be done.

Motor-cars were appearing of course, but were as yet far from reliable, and horses were still largely used. An old mate of mine, lately very ill in hospital, loved to talk about the old days, and was telling his nurse how Victoria Bridge was washed away in 1893. Her only comment was: "What happened to the motor-cars?" He was still chuckling about it all when I visited him later. In 1913 Dr. Jackson had no car and used to drive into town each morning in a rubber-wheeled sulky pulled by a grand little trotting pony called Rufus. Dr. Jackson did very little visiting, but now and then some special patient needed a visit and I was sent off in Rufus' care. I remember one very hot summer day, when I arranged two visits, one to Wooloowin and one to Moorooka. Both Rufus and I had had enough when we finished. We started off to Wooloowin down Queen Street; the sulky had no brakes and Rufus, with a hard mouth and a heart like a lion, hated having anything in front of him. He would catch up with any vehicle, pull out wide enough to see if there was room and then away we would go. His judgment was marvellous, and it was just as well, for there was little I could do about it!

Most maternity patients were delivered in their own homes, with a trained nurse. These nurses were, with very few exceptions, the salt of the earth—they took charge of the family, and nothing was left undone during their stay in the home. Nursing homes were of varied quality—those run by modern-trained nurses were excellent, but some, run by the old-fashioned "gamps", were far from being pleasant! Prenatal care, such as blood-pressure readings and urine tests, could only be classified as non-existent!

Gastro-enteritis amongst infants was prevalent and the death rate was very high. About the only way to feed a baby artificially was to boil and dilute cows' milk—a very poor substitute for mothers' milk—and rickets and infantile scurvy were very common. It was no wonder that everything possible was done to encourage the mother to persist with breast feeding, no matter what was happening to the recipient. Nowadays, I am informed, Dad, provided with a tin opener and a good bank balance, is in a much better position to cope with the situation than is Mum with her old-fashioned methods.

Apropos of infant feeding, and perhaps as an example of the modern trend to commercialism in medicine—a trend that we "old-timers" heartily deplore—the following little story might be of interest.

A medical student, in a recent year was asked in his exams to supply five reasons why mothers' milk should be preferred to any other substance for infant feeding. With great difficulty, he filled in the first three reasons with what scientific knowledge he had, and then he resorted to salesmanship. He filled in Number Four like this: "It is very convenient to take to the pictures, or to use while viewing television." The fifth reason he gave was so flagrantly of an advertising nature that I refrain from recording it.

I understand the student was of the Grimsdyke type, so popularized by Richard Gordon in his "Doctor" books.

In 1913 an incident of considerable scientific interest took place with reference to scurvy. A child was admitted to the Brisbane Children's Hospital with a very severe subperiosteal hæmorrhage, spongy gums and petechial hæmorrhages all over the body. Later a couple of less severe cases were admitted, all from the same address. At that time, it was the custom for the Government to farm out children to appropriate women and this was such a case. On investigation, it was found that this particular woman had been so successful that the number of children she was allowed to take had been increased recently. On further investigation, it was discovered that her former success had been due to the fact that she had kept goats and fed the children on plenty of goats' milk. For some reason, which I cannot remember, she had to get rid of the goats—and this tragic result had followed!

The approach to medicine generally was developing from the empirical to a more rational one, although preventive treatment was still a long way off. Diphtheria and typhoid were still prevalent, but antidiphtheritic serum was in use, and typhoid vaccine was showing remarkable results. One of the greatest troubles with

[1] The Jackson Lecture, delivered at the Annual General Meeting of the Queensland Branch of the British Medical Association on September 3, 1960.

diphtheria was the treatment of the quite numerous diphtheria carriers. It was a real problem, as they were isolated and had to have a succession of "negative" swabs—three, I believe— before being released from quarantine. Often, after two "negative" swabs, a positive would crop up, and the whole series had to start again.

Typhoid was treated mainly by starvation for three weeks or more. The patients became extremely wasted and often had bowel distress and even perforation. In 1914 I saw a whole ward of patients with typhoid, who were in fair condition after four or more weeks in bed, but still in a low febrile state. An increase in their diet reduced their temperature and then their recovery was uneventful. It was about this time that patients with typhoid were starting to be fed deliberately throughout their illness, with great benefit.

Tuberculosis was very common and the treatment was on general lines, with great emphasis on sunshine and climate.

Venereal disease was horribly common and very resistant to treatment. Gonorrhœa was treated mainly by local applications, and was very liable to develop complications, which were almost impossible to clear up. Syphilis was common enough, especially the tertiary variety. Treatment was really palliative only, as relapses were the rule rather than the exception.

The treatment of diabetes was a nightmare to doctor and patient alike, as regulation of diet was the only treatment, and the work which was entailed in calculating the right number of calories was enormous.

Kidney and bladder pathology belonged to the Dark Ages. Many times, in my medical-school days, I had listened to learned discourses about the nature of some kidney condition, and later, at the post-mortem examination, been shown some entirely different pathology. During the period from 1908 to 1913 at a big London teaching hospital, I can remember seeing or hearing of only one cystoscopy—and that was a failure, as the lamp would not work. Prostatectomy was only attempted as a last resort and the results were pitiful. On entering the ward of a hospital one could tell the presence of a "bladder case" by the prevailing ammoniacal smell. However, soon after that the cystoscope was perfected and urology developed with great rapidity.

Operations were still done, at times, in private homes. In 1901 my mother underwent a major pelvic operation in her own bedroom, and in 1903 I myself had an inguinal herniorrhaphy in the same room. The patient survived, the wound healed by first intention and the operation was successful. It still is, and the memory of the anæsthetic will never fade. I was given "closed ether" in a bag, and the induction was done, with ethyl chloride. To see an induction now is fantastic. However, anæsthetics were given by every Tom, Dick and Harry, and fatal results were very few and far between. It takes a very skilled operator to give a modern anæsthetic. From 1904 onwards, appendicectomies were being performed with increasing frequency, but the time to operate was still being debated. Rubber gloves were being used more and more frequently, but many surgeons used them only in the performance of an operation when gross infection was present—and then only to protect themselves.

Sepsis was still a menace—so much so that at Guy's between 1908 and 1912 there were two wards, called "septic wards", where suitable cases were isolated and kept apart from the rest of the hospital. The carbolic spray had disappeared from the operating room, and gradually asepsis was taking the place of antisepsis. Sir William Arbuthnot Lane had done a lot to promote the idea of asepsis by his brilliant technique in the plating of bones, and surgeons from all over the world were going to London just to see him handle his ingenious instruments—nothing went into the wound unless it had been boiled and nothing went through the skin except the knife, which was immediately discarded.

Diagnosis was based largely on common sense and experience. Treatment was also based largely on common sense and experience, with a more and more rational approach as physiology and pathology were developed. The use of X-ray films for diagnosis, of course, was becoming more widespread, but there were still plenty of unpleasant results, such as burns, from such usage.

In this atmosphere, Dr. E. Sandford Jackson was a commanding figure. He was a fine, upstanding man, with greying hair and dark, piercing eyes. He dressed well and carried himself well; he said very little when consulted, but expected his orders to be carried out explicity! He had a lovely home at Toowong, on the river bank, on several acres of land. He kept good riding horses on which he and a friend or two would exercise over the hills around Mt. Coot-tha. He also kept a motor-boat, with a man to run it, and on this he spent most weekends up or down the river. He kept no car, but had a grand trotting pony, as I said before, which took us into town every weekday. He operated four mornings a week, and saw patients the rest of the time at fixed hours. A taxi took him to and from lunch at the Queensland Club, where he always sat in the same chair at the same table. He was a great disciplinarian, and never expected to give an order twice. His work at the General Hospital, which I did not see, must have been of very great value to the community. His own hospital, St. Helen's, was a remarkably good hospital in 1913 and compared very favourably with London standards. He still used carbolic acid in the operating theatre, but the general cleanliness was excellent, and post-operative infective trouble was conspicuous by its absence.

If, by any chance, in my foregoing remarks I have given anyone the impression that I have a poor opinion of the Brisbane doctors of the 1900 to 1914 era, please let me correct that here and now. During 1913 and 1914 the beds of St. Helen's Hospital were filled entirely from Dr. Jackson's own practice. However, several of Brisbane's leading doctors appeared from time to time for some particular consultations. Amongst them I remember most Dr. Meek, Dr. Lilian Cooper, Dr. Lochhart Gibson and Dr. Turner.

Dr. Meek was a general practitioner of outstanding ability, both medical and surgical. He was unmarried, and was kindness itself to all his patients, and later on to many young doctors when they started in practice in Brisbane. In the early days his horses were famous, as were his motor-cars in later years.

Dr. Lilian Cooper was Brisbane's first lady doctor—and was one of the leading surgeons. I cannot recall Dr. Cooper without referring to Miss Bedford, the names of these two ladies being so often mentioned together. Miss Bedford was well loved and admired by all, and was very active in doing works for the public good, especially for the Children's Playground Association, for many years. In later years she drove a car rather fearsomely.

On one occasion she left it parked in Queen Street. When she returned she found a young policeman chalking her car for parking. She went up to him and said: "Young man, what are you doing, scribbling on my car? I don't like it." She then told him her name, and he meekly let her drive away without booking her. Later, on another occasion when she was driving her car rather erratically, a policeman asked her age. She replied: "Young man, no gentleman ever asks a lady her age." She won the day again, so well was she known, respected and liked.

Dr. Gibson was a leading ophthalmic surgeon, and Dr. A. J. Turner, whom I mentioned previously, a leading physician, with a great reputation for dealing with children's diseases. In earlier days, these two men had collaborated in research work concerning the causes of lead poisoning in children.

In those early days amongst students there was a definite idealism and devotion to duty, which the senior men carried on into their work amongst the sick and suffering. The good "oldtimers" were the men who interfered as little as possible with the patient and hoped Nature herself would make a cure (Nature did so in a surprising number of cases). Better still, these patients generally acquired some further immunity in the process. The modern expert has so many powerful, active treatments at hand—antibiotics, transfusions and innumerable

scientific tests—that it takes a very wise head to be sure of doing more good than harm. There is perhaps a far greater chance of the patient's surviving, I admit, but he often comes through his illness with a lowered resistance to that particular germ.

In one respect, the doctor in the early days had a great advantage over his modern counterpart. His patient trusted him, and anything the doctor had to say was accepted as fact. Nowadays a doctor is subjected to extreme criticism in all he says and does, and the patient, as often as not, reads all about the disease in the latest magazine before consulting him.

Recently a friend of mine had an experience that might be classed as an amusing example of the difference between modern and old-fashioned treatment. He was suffering from persistent abdominal pains and went to his doctor, who gave him 100 sulpha tablets, and diagnosed his case as a virus infection. However, his elder brother had had "'flu", and had been given 100 sulpha tablets, and had not enjoyed the experience. My friend therefore, after due deliberation, took a large dose of castor oil and went fishing for a couple of weeks. The treatment was 100% effective.

The advances in surgery and medicine had proceeded so dramatically since 1900 that the absence of a medical school was sorely felt in Brisbane, and it was very much to the credit of Dr. Jackson and his colleagues that they worked so hard for the establishment of one. The personality of Dr. Jackson and his professional standing in the community were of the greatest help in achieving this object.

After World War I there was an influx of young, keen, capable, modern-trained doctors, and the whole aspect of medical Brisbane changed. Dr. Jackson acquired a place at Victoria Point and absented himself more and more from St. Helen's, finally devoting himself to working for the establishment of a medical school, and delving into the early history of Brisbane. The medical school opened in 1935, and Dr. Jackson passed away in St. Helen's in 1938.

RECENT CONCEPTS IN THE ÆTIOLOGY OF PREECLAMPSIA.[1]

By E. V. MACKAY, D.G.O., F.R.C.S.E., F.R.A.C.S., M.R.C.O.G.,

Department of Obstetrics and Gynæcology, University of Melbourne.

ALTHOUGH the fundamental factor (or factors) responsible for preeclampsia remains obscure, appreciable advances have been made in the last few years in furthering our knowledge of this condition. These recent findings have indicated a diffuseness of ætiology rather than a single clear concept of the disease. Apart from this seeming multiplicity of causative factors, there are several difficulties in the way of progress at the moment. The major problem lies in the differentiation of cause and effect. For example, it is well known that utero-placental and renal blood flows are reduced in pregnancy toxæmia, but it is difficult to decide whether this is merely a secondary result of a prevailing vascular hypertonicity, or the major factor, with release of secondary mechanisms either humoral or nervous, to restore the organ homœostasis; there is evidence that both the above-mentioned organs can act in this way as a result of diminished blood flow. A further problem concerns the frequently irreconcilable results obtained by different investigators. This disparity of results will no doubt be solved with time. Some of the more specific ætiological factors will now be discussed.

Constitution.

There is no doubt that there is a strong predisposition in the individual make-up of some patients that renders them more liable to this condition than one would expect. It has often been observed that the incidence in relatives is usually higher than in the general population. Katu and Nagata (1959) in Japan, studying different family lineages, have noted the importance of hypertension and vascular accidents in the history. These workers found that the incidence of toxæmia in patients with hypertensive lineages was 34%, compared with 8% in those with a negative family history. These figures became more significant if allergic and angiospastic tendencies were also taken into account.

Another aspect of constitutional predisposition to receive attention is the androgyny score. This is a ratio of the bisacromial to the bisiliac diameter, and Rey and Coppin (1959) have reported that patients with high scores—that is, with a tendency to maleness—are more likely to develop pregnancy toxæmia.

In addition to these physical factors, the mental outlook of the pregnant patient may also be important. Soichet (1959) came to the conclusion that preeclampsia was more common when the patient's general attitude to men or to the pregnancy was one of rejection. Other factors, such as hostile environment, were also important. More work needs to be done to discover why these patients are at greater risk. It may be bound up with compensatory dietetic excesses, alteration of a general state of tension in the body and so on.

Nutritional Factors.

From a practical point of view, the attack in this direction has yielded the most significant results in lowering the severity, and to a lesser extent the incidence, of toxæmia of pregnancy. The pioneer work of Dixon Hughes and Hamlin in Sydney is well known, and there has been a considerable improvement in the figures for this disease where the high-protein, high-vitamin, low-carbohydrate, low-salt regime has been carried out. However, the results from some centres leave many questions unanswered. It is difficult to believe, in view of the experiences in the war years in Europe with drastically curtailed proteins and vitamins, that lack of these factors could be of significance in the ætiology. As far as salt is concerned, we have often been advised to regard it as akin to a poison in pregnancy (Best, 1957). Mulé, Tatum and Sawyer (1957) were the first to cast some doubt on this when they noted ill effects in toxæmic patients on a salt-poor diet. Robinson (1958) took the matter further and gave a large series of patients either a salt-rich or a salt-poor diet. The incidence of toxæmia, fœtal loss and accidental ante-partum hæmorrhage was much reduced in the group freely partaking of sodium chloride. Furthermore, salt when administered to patients with preeclampsia produced a rapid amelioration in signs. On the basis of these findings, it would seem that excess carbohydrate is the most likely dietetic factor. It has been postulated that glycogen deposition is associated with excess water retention, explaining the rapid gain in weight observed in some patients.

Another factor in the diet to receive mention is the quantity of roughage. Howeler and Hewson (1951) have reported a significantly increased incidence of toxæmia in patients whose diet contained decreased amounts of fibre.

Endocrine Factors.

Although an endocrine basis has been postulated as a cause of the disease, there is little evidence that either excess or lack of any hormone can be implicated. Œstrogens and progesterone are the hormones produced in largest amounts in pregnancy. There is some evidence that progesterone therapy can reduce the incidence of toxæmia if given early in the pregnancy for certain specific symptoms (Dalton, 1957). However, if the preeclampsia is established, this therapy is ineffective. If there is a basis for this treatment, it may involve the known naturetic effect of progesterone, or be related to a spasmolytic effect on either the uterus or blood vessels. Both œstrogen and progesterone lack are classically seen in cases of habitual

[1] Part of a symposium on "Toxæmias of Pregnancy", arranged by the Victorian State Committee of the Royal College of Obstetricians and Gynæcologists, and held at a meeting of the Victorian Branch of the British Medical Association on July 6, 1960.

abortion and in early idiopathic placental insufficiency; yet preeclampsia is not common in these conditions.

Adrenal cortical hormone in excess produces Cushing's syndrome, to which pregnancy toxæmia has been likened. However, recent work suggests that the excess hormone in the blood is merely a reflection of the increased amounts of the corticoid-binding protein, transcortin. In any case, pregnancy toxæmia is unlike this syndrome in many respects. Aldosterone, the main sodium-retaining steroid of the adrenal cortex, is usually present in decreased amounts in the urine in pregnancy toxæmia (Kumar *et alii*, 1959), raised levels of excretion in the urine chiefly reflecting the degree of dietary sodium restriction. In addition, preeclampsia is not classically seen in conditions in which large amounts of aldosterone are being secreted—as, for example, when pregnancy is complicated by the nephrotic syndrome or cardiac failure.

The possibility of vasopressin being a major ætiological factor is suggested by the work of McGillivray and Buchanan (1958), who found that the fluid retention in toxæmia was primarily a pure water phenomenon. Although its vasopressor and antidiuretic properties would theoretically explain the pathogenesis of the condition, plasma levels of this hormone have been reported to be normal in preeclampsia, and the occasional presence of toxæmia in diabetes insipidus would also make one cautious. There appears to be some controversy over the question of sodium chloride retention in preeclampsia, as both Dieckmann and Pottinger (1957) and Plentl and Gray (1959) consider that there is significant sodium retention in this disease. There seems little doubt that in the interior of the cell the sodium content is elevated (Kumar *et alii*, 1959).

The whole question of weight gain and its relationship to preeclampsia needs further study. Although Alexander and Downs (1953), Williams (1957) and many others have noted significant weight increases, it is not sufficiently appreciated, as the former workers have emphasized, that the great majority of patients with excess gain do not develop toxæmia. Fish *et alii* (1959), in a later study, found very little significance in the different weight gains of normal and toxæmic patients; and furthermore, the highest incidence of this disease was in those who gained less than 15 lb. over the whole pregnancy.

Renal Mechanisms.

The position of the kidney in relation to preeclampsia is still obscure. It is doubtful whether the methodology of renal experimentation is even yet sufficiently precise really to advance our appreciation of changes in pregnancy toxæmia. Recent reports of functional alterations in this disease have been at variance. As was mentioned earlier, the problem of primary and secondary effects is difficult to determine in this organ. Sophian's theory of reflex renal vasoconstriction in response to increased uterine tension has been challenged by Scott (1958) and others, largely on the basis of the absence of toxæmia in cases of gross hydramnios. However, one should note the difference between stretching and tension. Precise methods of measurement of uterine tension have indicated that the values obtained in hydramnios can be very variable, even if the fluid accumulation is considerable (Caldeyro-Barcia *et alii*, 1957). It is doubtful whether fully valid criticisms of this theory have yet been put forward. The fact that toxæmia is peculiar to pregnancy would perhaps indicate a secondary, but not necessarily an insignificant, role of the kidney in this disorder.

Utero-Placental Hæmodynamics.

Of considerable interest have been the findings of Bieniarz (1958, 1959) in relation to the position of the placenta in the uterus. This worker has noted that preeclampsia is present in 70% of cases in which the placenta is implanted high in the uterus, with a progressive diminution in incidence as lower segment sites are approached. It has been observed that the work load on the ovarian veins in high implantations of the placenta is considerable, the tension being increased several times with possible secondary effects on the kidneys. In support of this, Muresan and Ciobica (1959) have reported changes in the renal glomeruli of pregnant animals following ligation of the uterine veins; these changes consisted of swelling and ischæmia of the capillary loops and obliteration of the capsular space, presumably due to altered renal hæmodynamics.

It has already been mentioned that uterine or placental ischæmia may be involved in the causation of preeclampsia, there being both experimental and clinical evidence to support this theory. Mastboom (1948), working with pregnant bitches, was able to induce a significant rise of blood pressure when the aorta was temporarily clamped below the origin of the renal arteries. It has also been noted that preeclampsia is commoner in patients with hypoplasia of the aorta and iliac arteries, occurring in five out of eight patients with this condition (Clemetson, 1960). A further point in favour of this concept is the considerable disparity in the size of the uterine vascular tree between the nullipara and the parous woman (Horn, 1917), which is held to explain the higher incidence in the former group of patients. Although the placenta alone seems essential for the development of preeclampsia, there is often little to find when it is examined histologically. The most consistent change is a tendency to syncytial knot formation; an increase of true infarcts is not characteristic (Burstein *et alii*, 1957). Jeffcoate and Scott (1959), studying the placenta in Rhesus-immunized pregnancies, have found a correlation between the fœtal-placental weight ratio and the incidence of preeclamptic toxæmia. When this ratio approached unity, toxæmia was almost the rule. When the fœtus was severely affected, the placenta was shown to resemble in some ways that of a hydatidiform mole, another condition in which early toxæmia may occur. In both, œdema of the villi was present, with focal hyperplasia of the trophoblast. In addition, there was often an increase in the number of cells in the stroma of the villi. Although of significance, these findings are not characteristic of the commonly occurring toxæmia, and may well represent a different type of ætiology.

Release of Vasopressor Material.

Hunter and Howard (1960), in a recent study, have isolated a hitherto unknown substance from the decidua and amniotic fluid of toxæmic patients. This material has been called hysterotonin, and is distinct from angiotonin, the catechol amines, serotonin and vasopressin. It has distinct vasopressor activity and is also an oxytocic, although seemingly weaker in this activity than the natural hormone. It has no antidiuretic properties and is not found either in the placenta or in the membranes. Its formation is apparently very similar to that of angiotonin, the decidual enzyme in this case reacting with a globulin to produce the active material. Evidence of diminished amounts of the specific globulin fraction in preeclampsia is held to support this view.

Conclusion.

These, then, are some of the recent findings concerning the ætiology of this seemingly simple disturbance. There are obviously several well-substantiated theories of causation. Even now we know too little about its ætiology to distinguish clearly predisposing and precipitating factors. The influence of the placental site and the isolation of a vasopressor and oxytocic substance seem to have important implications for future study. It is obvious that a considerable amount of knowledge is accumulating, and it is probable that further significant advances will be made in the near future.

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TREATMENT OF TOXÆMIA OF PREGNANCY.[1]

By MARGARET A. MACKIE, M.B., B.S., D.G.O., M.R.C.O.G.,
Honorary In-Patient Obstetrician,
Royal Women's Hospital,
Melbourne.

TOXÆMIA of pregnancy may be classified into three main groups, as follows:

I. Acute toxæmia of pregnancy: (*a*) Preeclampsia—(i) mild, (ii) severe. (*b*) Eclampsia.

II. Chronic hypertensive vascular disease with pregnancy—(*a*) without superimposed acute toxæmia, (*b*) with superimposed acute toxæmia.

III. Unclassified toxæmia.

For the purposes of this discussion my remarks will be confined largely to Group I with brief reference only to Group II.

Diagnosis.

Mild preeclampsia should be diagnosed if, after 24 weeks' pregnancy, any two of the following three signs are present: (i) A rise of systolic blood pressure of 20 points, and/or of diastolic pressure of 10 points, over the initial blood pressure. This is the relative rise in blood pressure, to the importance of which our attention was first drawn by our Sydney colleagues Dixon Hughes and Hamlin. (ii) Generalized œdema, usually betrayed by excessive weight gain. (iii) Albuminuria of any degree.

There is considerable discussion as to the actual level of blood pressure which should be considered abnormal in a pregnant woman. One errs on the side of safety in taking a systolic blood pressure of 130 mm. of mercury, and a diastolic pressure of 80 mm. mercury at the initial visit as being the upper limit of normality. However, in diagnosis of the onset of preeclampsia it is the relevant rise that is important. A rise to 120/80 mm. of mercury in a woman whose initial reading was 100/70 mm. of mercury is just as important as a rise to 150/90 mm. of mercury in a woman whose initial reading was 130/80 mm. of mercury.

Severe preeclampsia is considered to have developed if the following criteria are evident: (i) at rest in bed, the systolic blood pressure is 160 mm. of mercury, and the diastolic pressure is 110 mm. of mercury or more. (ii) albuminuria of "one-third solid" or more is present; (iii) the urine volume for a twenty-four-hour period is less than 30 ml. per hour; (iv) cerebral or visual symptoms occur (in fact, symptoms of any kind other than œdema of the limbs). These severe signs and symptoms mean impending eclampsia, and as such need close supervision. Eclampsia, of course, means severe preeclampsia with fits.

Treatment of Preeclampsia.

The successful modern management of preeclampsia depends on the recognition of, and immediate action on, the early signs of this condition.

Weight gain and œdema are usually the earliest signs of preeclampsia, and although their presence does not always mean that preeclampsia is developing, the patient should be treated as though it is. A weight gain of more than one pound per week, or the presence of clinically evident œdema of the legs or hands, should demand an increase in the amount of rest. The patient should be instructed to spend at least 10 hours in bed in each 24 hours. The occurrence of continued excessive weight gain, or œdema, means that this rest period should be increased correspondingly. In addition, the patient should be instructed to reduce her salt intake, and her dietetic intake as a whole should be checked and carbohydrate reduced. Ammonium chloride, in a dosage of 15 grains three times daily may be given for four days, then discontinued for three days, and the course repeated. Other diuretics such as chlorothiazide are contraindicated, as their use removes the early warning signs without altering the course of a disease which, if neglected, can cause the death of the fœtus and threaten the life of the mother.

If, in spite of these measures, excessive weight gain continues with or without a rise in blood pressure, the patient should be confined to bed, and allowed up for toilet purposes only. This may be done safely at home if the rise in blood pressure is only minimal—namely, 15 to 20 mm. of mercury systolic pressure, and 5 to 10 mm. of mercury diastolic pressure above the initial reading. However, the patient should be examined at least twice weekly, and urine specimens tested for albumin at frequent intervals.

[1] Part of a symposium on "Toxæmias of Pregnancy", arranged by the Victorian State Committee of the Royal College of Obstetricians and Gynæcologists, and held at a meeting of the Victorian Branch of the British Medical Association on July 6, 1960.

If the weight gain continues and the blood pressure rises further—that is, if the systolic pressure rises more than 20 mm. of mercury or the diastolic pressure more than 10 mm. of mercury above the initial recording, or if albuminuria occurs, the patient should be admitted to hospital.

It should be realized, and cannot be repeated too often, that albuminuria is a late sign of preeclampsia, and that a patient showing any albumin in a catheter specimen of urine should be in hospital, unless the albuminuria is due to a urinary infection and is not associated with other signs of preeclampsia.

In hospital, such a patient should be confined strictly to bed for a few days. A mild sedative such as phenobarbital, or "Amytal", 32 mg., should be given three times daily, and a sedative should be prescribed at night. The diet should be poor in salt and rich in protein and vitamins. The blood pressure should be recorded at least twice daily, the fluid intake and output should be recorded, and every specimen of urine which is passed should be tested for the presence of albumin. The patient should be weighed every day on the same scale, and the weight recorded. The nursing staff should be instructed to report the development of any symptoms such as headache, eye signs or epigastric pain. Frequently weight loss is considerable and dramatic, œdema diminishes or disappears, urinary output rises, blood pressure falls and albuminuria disappears. The patient may then be allowed out of bed for increasing periods.

If albuminuria has not been present at any stage, and the blood pressure remains stable and weight gain is controlled, this patient may be allowed to go home. However, she will need supervision at least once a week and frequent urine tests until delivery. She is suffering from a condition which will be cured only when the pregnancy comes to an end.

If albuminuria has been present, the patient should remain in hospital until delivery, even if the urine becomes and remains clear for several days. Any increase in activity beyond a very limited degree will cause the rapid recurrence of albuminuria, sometimes with severe symptoms of fulminating preeclampsia.

Even with care, recurrence of albuminuria is almost inevitable in these patients, and when it does recur, and if it persists for several days, induction of labour is indicated, despite the period of pregnancy. If the pregnancy is carried on too long in the presence of albuminuria, risk of intrauterine fœtal death, accidental hæmorrhage or eclampsia is considerable.

On the other hand, if weight gain is not controlled and blood pressure does not fall to normal after a period of rest in bed in hospital, the patient should not be allowed to leave hospital, and activity should be restricted sufficiently to keep the blood pressure at that patient's minimal level. Hypotensive drugs are usually of little value in these patients, and again mask a valuable sign. The aim of treatment is to allow the pregnancy to continue to the end of the thirty-seventh week, when labour can be induced by artificial rupture of the membranes without great risk to the fœtus. If the patient is allowed out of hospital and away from strict supervision, the risk of rapid deterioration demanding earlier induction of labour is very great.

Severe Preeclampsia.

If, when first recognized, the condition has become one of severe preeclampsia with a blood pressure of 160/100 mm. of mercury or more, albuminuria "one-third" or more and pronounced œdema, urgent admission to hospital is necessary, as rapid development of fulminating preeclampsia with further rise in blood pressure, increasing albuminuria and symptoms of impending eclampsia is to be expected. These patients should be in a single room with a special nurse, and should be kept quiet, with adequate sedative to achieve this. The blood pressure should be estimated every four hours, and urinary output measured and all specimens tested for albumin.

Some improvement can be expected; but albuminuria rarely disappears in these cases, and the blood pressure rarely returns to normal. If improvement is satisfactory, an effort can be made to carry the pregnancy on to 36 or 38 weeks. However, more commonly improvement is only slight. In this case the pregnancy should be terminated when maximum improvement is obtained, usually at the end of about 48 hours.

If, however, there is no improvement in 24 to 36 hours, or if the condition worsens and eclampsia is impending, induction of labour by artificial rupture of the membranes should be carried out, whatever the degree of maturity of the fœtus. In severe cases, if labour does not follow within a reasonable time, Caesarean section may be necessary; but this is uncommon, as these patients usually come into labour without great delay.

Eclampsia.

Should eclampsia develop, an injection of 16 mg. of morphine should be given at once, followed by an intramuscular injection of 10 ml. of a 50% solution of magnesium sulphate. A further 2 ml. of magnesium sulphate solution is given every four hours, or if there is another convulsive seizure. No further morphine should be given at any stage, and not more than 20 ml. of magnesium sulphate should be given in 24 hours, as both are respiratory depressants. For this reason the respiratory rate should be checked and the knee jerks elicited before each injection of magnesium sulphate is given, and the injection withheld if the knee jerks are absent, or if the respiratory rate falls below 10 per minute. The antidote to overdosage of magnesium sulphate is calcium, and 10 ml. of a 10% solution of calcium gluconate for intravenous injection should be available always when concentrated magnesium sulphate solution is being used. In addition, the patient should be kept quiet by the administration of sedatives such as sodium phenobarbital (0·2 gramme by intramuscular injection every 6 to 8 hours). This may be alternated with paraldehyde (5 to 10 ml. by intramuscular injection) if the patient is very restless, or if the fits are difficult to control. Oxygen, a mouth gag and some suction apparatus must be available for use during a fit.

If the systolic blood pressure is over 180 mm. of mercury, hypotensive drugs may be used to avoid the risk of a cerebro-vascular accident and to prevent the recurrence of fits. However, too great a fall of blood pressure may jeopardize the urinary function by reducing the renal blood flow, and threaten the life of the fœtus by reduction of the placental circulation.

Induction of labour by artificial rupture of the membranes should be carried out when eclampsia is controlled, usually 12 to 24 hours after the last fit. Cæsarean section is rarely necessary.

Chronic Hypertensive Vascular Disease with Pregnancy.

In Group II, I shall first discuss chronic hypertensive vascular disease in pregnancy without superimposed toxæmia. Any patient whose blood pressure is 140/90 mm. of mercury or more on two occasions before the twentieth week of pregnancy should be regarded as belonging to this category unless signs of chronic nephritis are present.

Pregnancy usually progresses normally. In favourable cases the blood pressure falls in the middle trimester, but may rise later in the pregnancy.

Throughout pregnancy the patient should rest more than the average patient. She should always have an afternoon rest, and this should increase towards the end of the pregnancy. If the blood pressure is persistently 150/100 mm. of mercury or more, hypotensive drugs may be used. "Serpasil" in a dosage of 0·25 mg. three times a day is usually adequate, and may need to be reduced in the middle trimester.

If the systolic blood pressue is over 160 mm. of mercury or the diastolic pressure over 100 mm. of mercury, the patient should be admitted to hospital two

or three weeks before her due date, and labour should be induced about 10 days before the expected date of confinement.

Approximately one-third of these patients develop a superimposed toxæmia with a sudden rise in blood pressure, generalized œdema and albuminuria. The risk of accidental hæmorrhage and of intrauterine fœtal death is considerably increased in these cases. Induction of labour should be carried out at thirty-seven weeks' gestation, or earlier, if albuminuria persists despite treatment.

Conclusion.

Professor R. J. Kellar of Edinburgh has summarized the factors leading to failure in the treatment of toxæmia, and these are worth remembering: (i) Failure of the patient to report urgent symptoms. (ii) Failure on the part of the doctor to see the patient often enough. (iii) The speed with which the preeclamptic state may develop.

The second of these factors is our responsibility, and the more honestly we observe it, and the more meticulous our ante-natal care, the less frequently will the first and third factors be of importance.

PREECLAMPSIA AND ECLAMPSIA — RESULTS.[1]

By LANCE TOWNSEND,
Professor of Obstetrics and Gynæcology, University of Melbourne.

MY TASK in this Symposium is to present our results. I will first present statistics from the Royal Women's Hospital, Melbourne, then the incidence of eclampsia and preeclampsia in the maternal deaths occurring in Victoria, and finally a survey of the part ante-natal care played in the incidence and perinatal mortality of preeclampsia at the Royal Women's Hospital during 1959.

ROYAL WOMEN'S HOSPITAL STATISTICS.

The statistics concerning eclampsia will be presented first and will be followed by those relating to preeclampsia.

Eclampsia.

Reliable statistics are available from 1939. It is thus possible to present the figures for the last two decades. For convenience, yearly statistics will be presented at five-year intervals—namely, for 1939, 1944, 1949, 1954 and 1959.

The results for booked patients will be presented first, and these will be followed by figures referring to all patients. (A booked patient is one who has made at least two ante-natal visits to the hospital before admission.) The results derived from our booked cases indicate the efficacy of the methods of treatment used at that time, as far as prophylaxis and cure are concerned. Those figures which apply to non-booked patients will indicate the incidence and severity of eclampsia in suburban private practice.

Booked Patients.

Table I presents the incidence of eclampsia with the maternal and perinatal mortality. It should be noted that our present methods of treatment were first instituted in 1951, so that the years 1954 and 1959 may be compared with the previous years.

Figure I demonstrates that there was no real fall in the incidence of eclampsia until 1959, the 1944 and 1954 rates being nearly the same. However, there is a quite definite fall in the perinatal mortality in eclampsia, due in the main to the reduction in severity and to the fact that a greater percentage of eclampsia in 1954 and in 1959 occurred *post partum*.

[1] Part of a symposium on "Toxæmias of Pregnancy", arranged by the Victorian State Committee of the Royal College of Obstetricians and Gynæcologists, and held at a meeting of the Victorian Branch of the British Medical Association on July 6, 1960.

All Patients.

Table II presents our total experience in all patients treated at the hospital. Figure II illustrates that the incidence has been falling since 1944, and tha this

TABLE I.
Eclampsia: Royal Women's Hospital Booked Patients.

Year.	Number of Deliveries.	Number of Patients with Eclampsia.	Maternal Mortality.	Perinatal Mortality.
1939	3099	20	3	7
1944	2931	6	0	6
1949	5377	17	0	1
1954	6622	12	0	1
1959	7180	5	0	1

improvement was initiated before the present methods of treatment were instituted in 1951. Figure III shows the maternal mortality. It is noteworthy that there were no

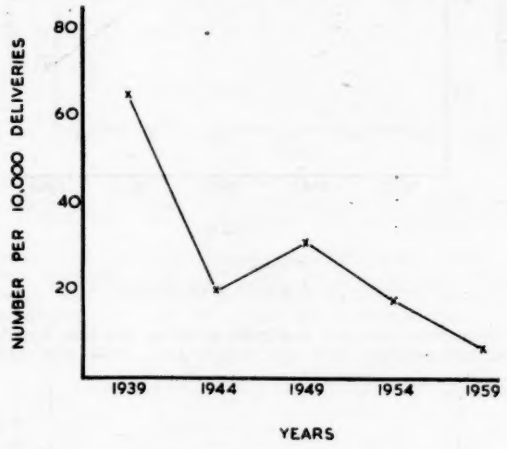

FIGURE I.
Eclampsia; incidence in booked patients.

maternal deaths in 1954 and 1959; this substantiates the previous statement that the disease is not as severe as it was. The graph also suggests that it may be no use drawing

TABLE II.
Eclampsia: Royal Women's Hospital, All Patients.

Year.	Number of Deliveries.	Number of Patients.	Maternal Mortality.	Perinatal Mortality.
1939	3743	42	8	15
1944	3583	39	1	14
1949	5865	45	6	8
1954	6622	26	0	6
1959	7721	18	0	7

conclusions from a statistical analysis, since in 1944 there was only one maternal death from eclampsia at the hospital. This year was unique, the death rate being high in 1943 and again in 1945. The perinatal mortality for all patients has fallen over the two decades, but it must be noted that the improvement since 1949 has been small.

To sum up: (i) There has been a gradual reduction in the incidence of eclampsia since 1944. (ii) The disease has become less severe, as shown by a fall in both the maternal and the perinatal mortality figures.

Preeclampsia.

The classification and definition of preeclampsia have changed in the two decades under review. Statistics

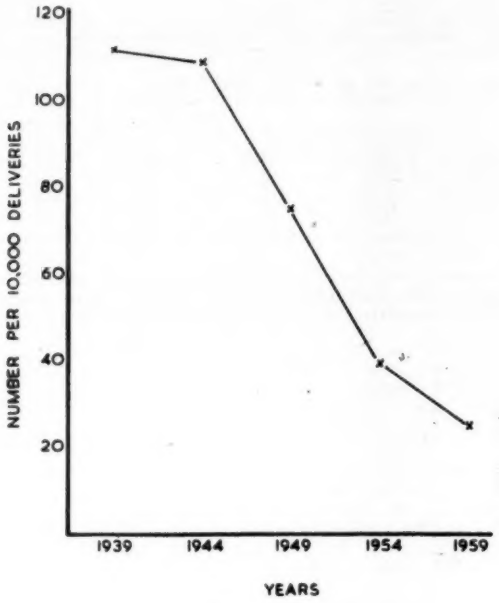

Figure II.
Eclampsia; incidence in total admissions.

for comparison are not available save for the last decade, so in consequence only the years 1949, 1954 and 1959

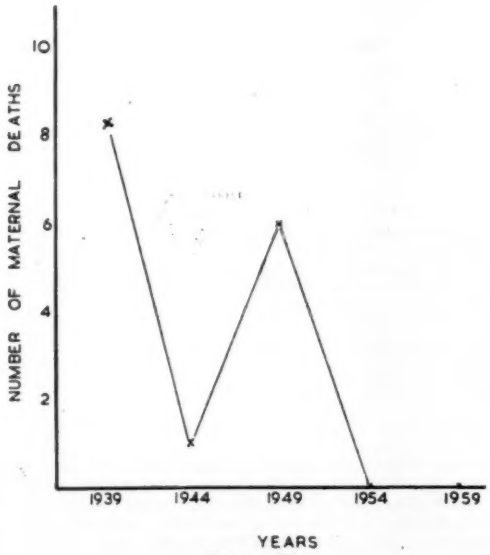

Figure III.
Eclampsia; maternal deaths.

will be presented. The year 1949 will serve as a control for the latter years.

Booked Patients.

Table III shows the incidence of preeclampsia in booked patients and the corresponding perinatal mortality. Figure IV indicates that the incidence rose in 1954 and returned to its previous level in 1959. Figure

Table III.
Preeclampsia: Royal Women's Hospital, Booked Patients.

Year.	Number of Deliveries.	Number of Patients with Preeclampsia.	Perinatal Mortality.
1949	5377	416 (7·5%)	18 (4·3%)
1954	6622	710 (10·7%)	29 (4·1%)
1959	7180	618 (1·6%)	29 (4·5%)

V reveals that the perinatal mortality has not changed in the decade under review. However, although it is not seen in the statistics presented, there was a tendency in 1959 for the severity of the preeclampsia to

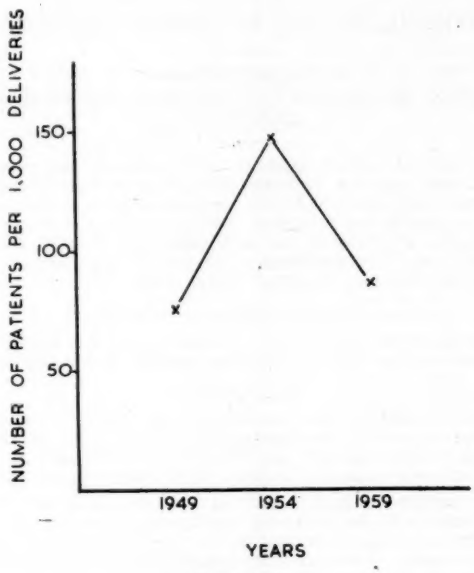

Figure IV.
Preeclampsia; incidence in booked patients.

have become less and the time at which it occurred in pregnancy to have become later than in 1949. It was disappointing that the over-all incidence had not changed and the perinatal mortality was stationary.

Maternal Mortality in Victoria.

Since 1953, all maternal deaths in Victoria have been investigated by a committee set up by the Minister of Health. Reliable statistics are available, and an accurate statement can be made concerning any death in which the patient had either preeclampsia or eclampsia. Figures VI and VII illustrate all maternal deaths and those associated with preeclampsia and eclampsia. In the years 1953, 1954 and 1955, the total number of deaths associated with the two diseases was stationary. In 1956 there was at first a substantial fall, and then a slight rise to a stationary level in 1957, 1958 and 1959.

These statistics show that, over the period concerned, there has been a fall in the maternal deaths associated with eclampsia and preeclampsia, and that over the

TABLE IV.
Royal Women's Hospital, 1959.

Period.	Total Number of Patients.	Mild Pre-eclampsia.	Severe Pre-eclampsia.	Eclampsia.	Total Pre-eclampsia and Eclampsia.	Total Perinatal Mortality.	Perinatal Deaths in which Preeclampsia was a Factor.
Before 20 weeks	4716	166 (3·5%)	75 (1·6%)	7 (0·15%)	248 (5·2%)	145 (3·1%)	10 (0·2%)
After 20 weeks	3190	92 (2·9%)	89 (2·8%)	10 (0·31%)	191 (6·0%)	200 (6·2%)	28 (0·9%)

last three years 10% of all maternal deaths in Victoria have been those of patients with this disease. In 1953 this figure was 15%. Finally, there has been only one maternal death from eclampsia in Victoria in the last three years, among more than 60,000 deliveries *per annum*.

ROYAL WOMEN'S HOSPITAL, 1959.

Dr. I. Johnson of my department carried out a survey in booked patients delivered at the hospital in 1959 to ascertain the value of our treatment of preeclampsia. Preeclampsia was defined as a condition in which any two of the following three criteria were present (*a*) generalized œdema; (*b*) a relative rise in blood pressure

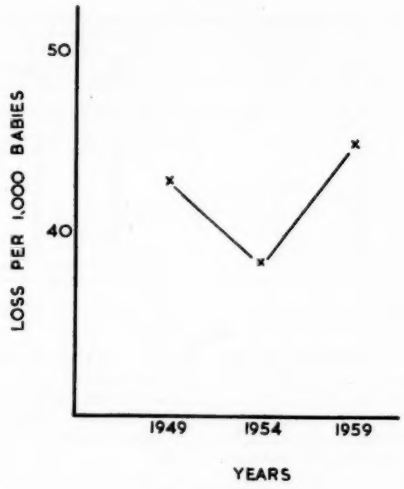

FIGURE V.
Perinatal mortality.

of over 20 mm. of mercury, systolic, or over 10 mm., diastolic; (*c*) proteinuria. Table IV shows the results of the survey. Severe preeclampsia was said to be present when a systolic blood pressure of 170 mm. or more was found, or when the protein content after the urine had settled constituted half or more of the whole volume tested.

Patients were placed in two groups — those who presented before 20 weeks and those who presented themselves after this time. As multigravidæ tend to present themselves later in pregnancy than primigravidæ, there were more primigravidæ and multigravidæ with a bad obstetrical history in the group presenting before 20 weeks than in the group presenting after 20 weeks. The bias would thus be against the group presenting before 20 weeks, as preeclampsia would be more common in this group.

When the two groups are compared, the following observations emerge: (i) The total incidence of eclampsia and preeclampsia in each is the same. (ii) The incidence of eclampsia was lower in the group who presented themselves before 20 weeks. (iii) The incidence of severe preeclampsia was similarly lower. (iv) The incidence of mild preeclampsia was substantially greater in the

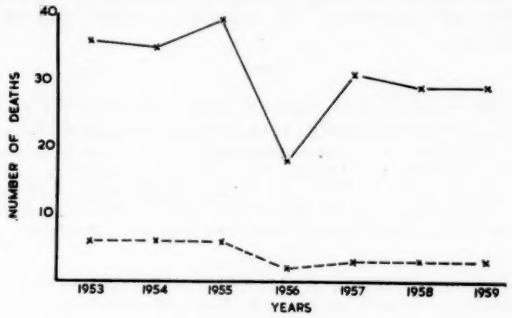

FIGURE VI.
Victoria—maternal deaths. Interrupted line, deaths due to eclampsia and preeclampsia; solid line, total deaths.

group who presented themselves before 20 weeks. (v) The perinatal mortality due to preeclampsia in the group who presented themselves before 20 weeks was one-quarter that of the group who presented themselves

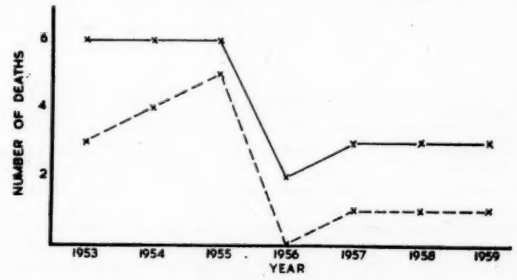

FIGURE VII.
Victoria — deaths due to eclampsia and preeclampsia. Solid line, total deaths; interrupted line, deaths due to eclampsia.

after 20 weeks. It should also be noted that the over-all perinatal mortality from all causes in the group who presented after 20 weeks was twice that of the group who presented before 20 weeks.

This survey shows that there is a reduction in the severity of the disease in the mother and a reduction in the perinatal mortality when ante-natal care is instituted early in pregnancy.

CONCLUSIONS.

From the data presented, the following conclusions may be reached concerning present-day methods of treatment of preeclampsia and eclampsia.

1. The incidence of eclampsia has been reduced.

2. The severity of eclampsia is less, as judged by a fall in maternal and perinatal mortality.

3. The same remarks apply to severe preeclampsia.

4. When ante-natal care is instituted before 20 weeks, the incidence of eclampsia and of severe preeclampsia is less than when such care is commenced after this time.

5. Modern methods do not reduce the over-all incidence of preeclampsia. They reduce the severity of the disease and delay its onset.

6. The time has come to analyse critically our current methods of treatment and to assess the relative values of attendance for ante-natal care, high-protein reduction diet, salt restriction, diuretics and bed rest. Some or all of these methods of treatment have produced the above-mentioned results. I predict that the important one will turn out to be the making of regular visits to the doctor, so that a clinical examination can be made and admission to hospital ordered whenever necessary.

THE ANTIBIOTIC SENSITIVITY OF STAPHYLOCOCCUS AUREUS IN THE BRISBANE HOSPITAL DURING 1959.

By J. F. R. KERR, M.B., B.S., B.Sc.,
Pathology Department, Brisbane Hospital, Brisbane.

WHEN a medical officer is faced with the problem of treating a patient in hospital with an infection which, he suspects, is caused by *Staphylococcus aureus*, three important factors will influence his choice of antibiotic. First, he will have formed an opinion, based on the results of sensitivity tests he has seen recently, as to what antibiotic is most likely to be successful. This opinion will be based on the results of a small number of cultures, and will not always be accurate. Secondly, he will endeavour to refrain from using those antibiotics, if any, which have been set aside for the treatment of severe, resistant staphylococcal infections in the hospital concerned. Finally, he will assess the severity of the infection and decide whether one of these "guarded" antibiotics should be used before the results of sensitivity tests are available.

TABLE Ia.
Results of Sensitivity Tests of Staphylococcus Aureus (In-Patients, 1959).

Antibiotic.	Resistant Strains.	Sensitive Strains.	Total.
Penicillin	929 (86%)	152 (14%)	1081
Streptomycin	204 (19%)	877 (81%)	1081
Chloramphenicol	17(1·6%)	1064 (98·4%)	1081
Tetracycline and/or oxytetracycline	454 (42%)	627 (58%)	1081
Erythromycin	8 (0·7%)	1073 (99·3%)	1081

This report sets out the antibiotic sensitivities of coagulase-positive, pigment-producing strains of *Staph. aureus*, isolated from pus and sputum at the Brisbane Hospital during 1959. It is hoped that the figures will provide a useful guide to therapy, and that they will be of interest in that they demonstrate the result of the exercise of restraint in the use of erythromycin in this hospital.

Methods.

The antibiotic sensitivities of the organisms were assessed after 18 hours' incubation on nutrient agar. When the primary culture yielded several species of bacteria, subcultures of the various species were carried out to determine the antibiotic sensitivity of each.

Evans "Sentest" tablets of the following strengths were used: penicillin, 2·5 international units; streptomycin, 80 γ; chloramphenicol, 100 γ; erythromycin, 10 γ; tetracycline, 100 γ, oxytetracycline, 100 γ.

Results.

The results of sensitivity tests, performed on organisms isolated from in-patients and from patients attending the casualty department, are considered separately. The results of cultures obtained from patients attending the out-patient department are excluded from this discussion, as many of these patients would have been recently discharged from the hospital. The majority of the swabs received from the casualty department were taken from furuncles, carbuncles, hand infections and small abscesses. The casualty department in this hospital is not used for the follow-up of patients recently discharged from the wards, and the sensitivities of organisms obtained from this department should give a reliable indication of conditions pertaining in general practice.

TABLE Ib.
Results of Sensitivity Tests of Staphylococcus Aureus (Casualty Patients, 1959).

Antibiotic.	Resistant Strains.	Sensitive Strains.	Total.
Penicillin	1021 (64%)	570 (36%)	1591
Streptomycin	21 (1·3%)	1570 (98·7%)	1591
Chloramphenicol	0 (0·0%)	1591 (100%)	1591
Tetracyclines	105 (6·6%)	1486 (93·4%)	1591
Erythromycin	3 (0·2%)	1588 (99·8%)	1591

TABLE II.
Sensitivities of Streptomycin-Resistant Staphylococci (In-Patients, 1959).

Penicillin.	Streptomycin.	Chloramphenicol.	Tetracycline.	Oxytetracycline.	Erythromycin.	Number of Strains.
R	R	S	R	R	S	164
R	R	S	S	S	S	15
R	R	R	R	R	S	13
R	R	S	R	R	R	5
S	R	S	R	R	S	2
S	R	S	S	S	S	2
R	R	S	S	R	S	1
S	R	R	R	R	S	1
R	R	R	R	R	R	1
Total						204

TABLE III.
Sensitivities of Chloramphenicol-Resistant Staphylococci (In-Patients, 1959).

Penicillin.	Streptomycin.	Chloramphenicol.	Tetracycline.	Oxytetracycline.	Erythromycin.	Number of Strains.
R	R	R	R	R	S	13
R	S	R	R	R	S	1
R	R	R	R	R	R	1
R	S	R	S	S	S	1
S	R	R	R	R	S	1
Total						17

Table I shows the sensitivities of 1081 strains of *Staph. aureus* obtained from in-patients and 1591 strains obtained from casualty patients.

The origins of the 17 strains of staphylococci obtained from in-patients, found to be resistant to chloramphenicol, were as follows: wound infections, 4 strains; skin infections, 4 strains; sputa, 3 strains; ear infections, 2 strains; brain swab at autopsy, 1 strain; pleural effusion, 1 strain; tracheotomy, 1 strain; vaginal swab, 1 strain.

The origins of the eight strains of staphylococci obtained from in-patients, found to be resistant to erythromycin, were as follows: wound infections, 3 strains; ear infections, 2 strains; finger infection, 1 strain; empyema, 1 strain; skin infection, 1 strain.

TABLE IV.
Sensitivities of Staphylococci Resistant to the Tetracyclines (In-Patients, 1959).

Penicillin.	Strepto-mycin.	Chlor-amphenicol.	Tetra-cycline.	Oxy-tetra-cycline.	Erythro-mycin.	Number of Strains.
R	S	S	R	R	S	253
R	R	S	R	R	S	164
R	R	R	R	R	S	13
S	S	S	R	R	S	9
R	R	S	R	R	R	5
R	S	S	S	R	S	2
R	S	S	R	R	R	2
S	R	S	R	R	S	2
R	S	S	R	S	S	1
S	R	R	R	R	S	1
R	S	R	R	R	S	1
R	R	R	R	R	R	1
Total						454

Discussion.

The penicillin resistance of hospital staphylococci is well known. Only 36% of the casualty-department strains were sensitive to penicillin. This figure is not significantly less than the 43% of penicillin-sensitive strains found in 1618 cultures done in general practice throughout Australia in 1958 (Johnson *et alii*, 1960) where the same "Sentest" tablets were used.

TABLE V.
Sensitivities of Erythromycin-Resistant Staphylococci (In-Patients, 1959).

Penicillin.	Strepto-mycin.	Chlor-amphenicol.	Tetra-cycline.	Oxy-tetra-cycline.	Erythro-mycin.	Number of Strains.
R	R	S	R	R	R	5
R	S	S	R	R	R	2
R	R	R	R	R	R	1
Total						8

Nineteen *per centum* of strains from in-patients were resistant to streptomycin. Of these, 80% were resistant to tetracycline also. The common practice of using penicillin and streptomycin in combination for hospital patients, particularly after operations, may well have been responsible for the development of many of these streptomycin-resistant strains. Because of the rapid development of resistance to streptomycin by staphylococci, it is desirable to avoid its use for staphylococcal infections as a single agent when other antibiotics are available (Lowbury, 1960). The percentage of staphylococci obtained from the casualty department found to be resistant to streptomycin (1·3%) is almost identical with that found in the 1958 general-practice survey (1·4%) (Johnson *et alii*, 1960), and is significantly less than that found in staphylococci from in-patients.

Not one of the 1591 strains from the casualty department was resistant to chloramphenicol. Only 1·6% of the strains from in-patients were resistant. Of these 17 resistant strains, 13 were resistant to all the other antibiotics tested except erythromycin. The origins of these strains indicate that they were isolated from infections, most of which could have been acquired in hospital. Resistance to chloramphenicol develops slowly, but becomes high in hospitals where much chloramphenicol is used (Lowbury, 1960). The danger of a blood dyscrasia resulting from one restricted course of chloramphenicol is slight (Dunlop and Murdoch, 1960).

For some years tetracycline has been widely used in this hospital. It is still commonly used, even when the infection is thought to be staphylococcal and to have been acquired in the hospital. Forty-two *per centum* of the in-patient staphylococci were resistant to tetracycline. The percentage of resistant casualty-department strains (6·6%) was significantly less. Whereas the majority of the in-patient strains resistant to chloramphenicol and erythromycin were highly resistant strains, being resistant to most of the other antibiotics tested, 56% of those resistant to tetracycline were resistant to the tetracyclines and penicillin only. Nine strains were resistant to the tetracyclines alone.

TABLE VI.
Sensitivities of Streptomycin-Resistant Staphylococci (Casualty Patients, 1959).

Penicillin.	Strepto-mycin.	Chlor-amphenicol.	Tetra-cycline.	Oxy-tetra-cycline.	Erythro-mycin.	Number of Strains.
R	R	S	R	R	S	13
R	R	S	S	S	S	3
S	R	S	R	R	S	3
R	R	S	S	S	R	1
R	R	S	R	R	R	1
Total						21

TABLE VII.
Sensitivities of Staphylococci Resistant to the Tetracyclines (Casualty Patients, 1959).

Penicillin.	Strepto-mycin.	Chlor-amphenicol.	Tetra-cycline.	Oxy-tetra-cycline.	Erythro-mycin.	Number of Strains.
R	S	S	R	R	S	85
R	R	S	R	R	S	13
S	R	S	R	R	S	3
S	S	S	R	R	S	3
R	R	S	R	R	R	1
Total						105

TABLE VIII.
Sensitivities of Staphylococci Resistant to Erythromycin (Casualty Patients, 1959).

Penicillin.	Strepto-mycin.	Chlor-amphenicol.	Tetra-cycline.	Oxy-tetra-cycline.	Erythro-mycin.	Number of Strains.
R	S	S	S	S	R	1
R	R	S	S	S	R	1
R	R	S	R	R	R	1
Total						3

The incidences of resistance to erythromycin in cultures from in-patients and from casualty patients were low and did not differ significantly from one another. In this hospital, erythromycin has been reserved, as much as possible, for use in cases of severe infections by staphylococci resistant to other antibiotics. It has been shown that resistance to erythromycin develops readily and resistant strains become common in hospitals where much erythromycin is used (Lowbury, 1960). The absence of any significant difference between the incidences of erythromycin resistance in strains from in-patients and casualty patients indicates the success of the policy of

restriction of the use of this antibiotic in the prevention of the development of a large number of resistant hospital strains.

Summary.

The antibiotic sensitivities of strains of coagulase-positive *Staph. aureus*, isolated at the Brisbane Hospital during 1959, are tabulated.

The incidence of resistance of hospital strains to tetracycline was 42%. This is correlated with the widespread use of this antibiotic in the Brisbane Hospital during recent years. On the other hand, the frequency of in-patient strains resistant to chloramphenicol was low.

Erythromycin has been reserved for the treatment of resistant staphylococci in this hospital. The low incidence of erythromycin resistance in in-patient strains, and the known facility with which staphylococci develop resistance to this antibiotic, indicate the success of this policy.

Acknowledgements.

I am grateful to Dr. A. W. Pound, Director of the Department of Pathology, and to Dr. A. F. Knyvett, Acting General Medical Superintendent of the Brisbane Hospital, for permission to publish these figures. The cultures were performed by the staff of the bacteriology department of the Brisbane Hospital under the direction of Miss E. Wilson.

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Reviews.

Chemical Micromethods in Clinical Medicine. By R. H. Wilkinson, M.A., M.D.; 1960. Oxford: Blackwell Scientific Publications Ltd. 8½" × 5½", pp. 136, with illustrations. Price: 40s.

THIS book describes various microchemical methods in use in the Biochemistry Laboratories of the Hospital for Sick Children, Great Ormond Street, London. It is essentially a collection of recipes preceded by two useful chapters on the principles involved in applying micromethods to the examination of blood, on the collection of specimens from young children and on the organization of a biochemical laboratory.

Details of methods for 35 or so of the commonest biochemical estimations are given, together with an account of their use in the assessment of patients. Practically all these methods described are for use in the examination of blood. It is surprising to find that the examination of cerebro-spinal fluid is not considered, and while the importance of urinary examination is mentioned, few details of methods are given. Perhaps this is because micromethods are not so necessary in the examination of urine; yet, some urine tests are mentioned specifically, while with others the reader is left to find out elsewhere whether or not the methods given for blood examination are suitable for urine.

A recipe book, in our opinion, should contain sufficient detail to allow a trained laboratory technician to assemble the reagents and apparatus and get on with the job. While this is possible with many of the methods given, some unfortunately lack important details. Thus, in the description of a method for estimating plasma sodium content, it is stated that values for an unknown plasma sodium content are read off a calibration curve, yet no instructions are given for the construction of such a curve. Similarly, under the description of a micro-Kjeldahl method for estimation of plasma proteins (surely not a very popular method), the reader is instructed to calculate a non-protein nitrogen correction from the blood urea value, but is not told how to make the calculation. While such points may seem trifling to someone who has spent many years in a laboratory, they can be a source of great loss of time to a less experienced person setting up a method for the first time.

A useful table of normal biochemical values for children compared with those of adults is given in an appendix. Here again, however, there is room for improvement. Plasma calcium values are given in milliequivalents per litre, though the method described earlier gives results in milligrammes per 100 ml. Serum acid phosphatase values are given for adults, but not for children. Recent studies in Gaucher's disease suggest that this test may be important in relation to children also. Normal values are given for plasma fibrinogen content, yet a method for its estimation is not described. On the whole, this is a disappointing book.

Clinical Physiology. Edited by E. J. Moran Campbell, B.Sc., Ph.D., M.D., M.R.C.P., and C. J. Dickinson, B.A., B.Sc., B.M., M.R.C.P., with a foreword by Sir Robert Platt, Bt., M.D., M.Sc., LL.D.; Oxford: Blackwell Scientific Publications, Limited. 8¾" × 5½", pp. 542, with 29 figures and 26 tables. Price: 50s. (English).

THIS book was produced, according to the editors, because there are few good textbooks dealing with clinical physiology. Their aim was to fill this gap with a book written by clinicians, discussing those aspects of physiology best looked at from the clinical standpoint. The 15 chapters covering most bodily functions except those of the nervous system are each divided into four sections: normal function, disordered function, physiological basis of tests and measurements, and "practical assessment". The authors of the individual chapters are all English physicians (and one surgeon) of the younger post-war school.

Such a book may be, as Sir Robert Platt points out in his foreword, long overdue. The result is a little disappointing. As with most multi-author books, one of the main defects is unevenness among the various sections, both in quality and in quantity. Some chapters, such as Campbell's on respiration, are first-class. The chapter on the blood and bone marrow lies at the other end of the scale, the remainder being somewhere in between. Some sub-sections are frankly clinical, with all the hidden empiricism that this implies. One may also be a little unhappy about the balance of a textbook of "clinical physiology" which devotes 45 pages to the circulation, 58 to respiration, 97 to the gut and its appendages, and 145 to various aspects of endocrinology (including no less than 53 to "sex, reproduction and growth").

A more fundamental criticism could be aimed at the general achievement of the authors and editors. Despite the foreword and the preface, it is hard to see where this book "fits in". The better modern textbooks of medicine and physiology correlate the physiological basis of medicine and the medical aspects of physiology moderately well. The present work could be regarded as a generally useful book only if it connected the two disciplines superbly well. In all but a few chapters, it fails to do this.

It is difficult to see to which of the three groups suggested by Sir Robert Platt this book could best be recommended. For the student there are other and better sources; the recent graduate will usually seek a less superficial approach; and we are left only with "the established physician who realizes that the younger men have been getting ahead of him". In some chapters only will the last-mentioned find first-class help in bridging the gap.

Books Received.

"Medical Evidence in Personal Injury Cases: The Medico-Legal Aspects of an Action for Damages Arising out of a Personal Injury", by Dorothy K. Dix and A. H. Todd, M.S., B.Sc. Lond., F.R.C.S. Eng.; 1961. London: H. K. Lewis & Co. Ltd. 8½" × 5½", pp. 254 with a few illustrations Price: £2 2s. net (English).

"Thymectomy for Myasthenia Gravis: A Record of Experiences at the Massachusetts General Hospital", by H. R. Viets, M.D., and R. S. Schwab, M.D.; 1960. Illinois: Charles C. Thomas; and Oxford: Blackwell Scientific Publications Ltd. 9" × 6", pp. 160. Price: 56s. (English), with illustrations.

"Progress in Endocrinology: Part I: Neuroendocrinology and Endocrinology of the Thyroid and Parathyroid Glands", Memoirs of the Society for Endocrinology, No. 9, edited by K. Fotherby and J. A. Loraine, J. A. Strong and P. Eckstein; 1960. Cambridge University Press. 9¾" × 7", pp. 176, with a few illustrations. Price: 45s. net (English).

The Medical Journal of Australia

SATURDAY, APRIL 15, 1961.

THE CONSTITUTION OF THE AUSTRALIAN MEDICAL ASSOCIATION.

THE item of most far-reaching importance on the business paper of the Federal Council at its recent meeting (see page 564) was the constitution of the Australian Medical Association. Drafts of the Memorandum of Association and Articles of Association had been prepared by the Federal Council's legal advisers, and these, after careful examination and appropriate amendment, were approved and referred back to the Branches for their further consideration. It is intended then that they will come before a special meeting of the Federal Council, presumably early in June, which should leave time for the completion of the various necessary formalities, including submission to the Solicitor-General in Canberra, so that the new association can be brought into being by the beginning of 1962. The Australasian Medical Congress to be held in Adelaide in May, 1962, will then provide an excellent occasion for the holding of the inaugural meeting of the Australian Medical Association and the first meeting of the Federal Assembly.

This progress would not seem to have any undue haste about it. It has allowed time for fair consideration of the matter and yet conforms to seemly expedition. It is two years since the Federal Council, at its meeting in Sydney in March, 1959, resolved to take the necessary steps to investigate the possibility of formation of a medical association in Australia, recognizing the increasing call for and the inevitability of the formation of such an association. We commented at the time[1] that it was probably not too rash to predict that within three years the new association would be an actuality, and that is how it seems to be working out. At the next meeting of the Federal Council, at the end of September, 1959, the pros and cons[2] were considered. More positive steps were then taken with the passing of a resolution, described by the President as being of a general character, but expressing the Federal Council's considered opinion that the time was now opportune to proceed with the formation of an Australian Medical Association, independent of, but affiliated with, the British Medical Association. This opinion was then, of course, referred to the Branches with a request that the views of members be obtained, but the Federal Council's decision was momentous.

The six Branches of the Association then approached their members in the various ways customary in the individual States and in every case reported to the Federal Council approval of the formation of the new association. This is not the time and place to analyse the merits and demerits of the means adopted in the several Branches to obtain the opinions of their members on this or other questions, whether by reference to local associations, by convocations or by a plebiscite of members. However, it is interesting to note that in the one State, Queensland, in which members were approached for their individual views, circulars were sent to 1341 members, of whom 332 replied; 309 agreed with the suggestion, 15 disagreed, two were uncertain, and the replies of six were informal. The important point to the Federal Council, indeed, the only point that concerned it, was that the six Branches had approved the suggestion. It then set up a steering committee to do the serious job of making the idea a reality. This has been a major task, but it is now virtually over. Perhaps three over-riding considerations had always to be borne in mind: (i) the organizational, administrative and legal soundness of the constitution to be adopted; (ii) the incorporation in it, so far as was humanly possible, of the ideas and ideals of those who could be expected to make up its immediate future membership; (iii) the cultivation of unity in the whole profession. To this end the steering committee spent very many hours of 1960 pondering and planning, discussions by correspondence and in person were carried on with the Colleges and other groups within the profession, the detailed views of Branch Councils were obtained on a draft constitution, and at the end of November, 1960, the draft constitution was considered critically by a convention more fully representative of the medical profession than any previous gathering held in Australia. This was an historic occasion. Every ideal was not achieved, compromises were necessary, some minority views had to be put aside, but an important measure of agreement was obtained, even where it was agreement to differ. Whatever regrets anyone may have, the realistic view is surely that the whole matter was given thorough consideration and that little, if anything, more would be achieved by further discussion and delay.

The steering committee's task was then to sort out the recommendations of the convention and to submit the final essentials of the constitution to the Federal Council's legal advisers to be recast in appropriate legal form. The result was the bulky document before the Federal Council at its meeting, consisting of, first, the Memorandum of Association and, second, the Articles of Association. With minor amendments this has now been referred back to the Branches for their final comments. At this stage it seems reasonable to suggest that the matter has gone past the stage of major amendment. What is being submitted to the Branches is, in effect, the product of the convention cast in legal form. During last year's discussion every encouragement was given to the idea that the draft constitution should be "thoroughly kicked around". All that is regarded as appropriate now is the tying up of any loose ends or the removal of any serious errors that may have been overlooked. The whole matter has been fairly considered, and it will need all the time from the Federal

[1] MED. J. AUST., 1959, 1: 671 (May 16).
[2] MED. J. AUST., 1959, 2: 727 (November 14).

Council's special meeting in June to bring the new association into being by 1962. To those who would counsel further delay and reconsideration of major issues in the constitution two thoughts are suggested. The first is that the constitution, once adopted, is not immutable. It is true that the Memorandum of Association, which determines the name and contains the objects of the Association and certain other matters mainly to do with its income and property, cannot be easily changed, though it is not impossible to do so; but we are not aware of any important disagreement about the contents of the Memorandum, apart from the name of the association, which is set down as "Australian Medical Association". Those who have favoured the name "The Medical Association of Australia" and have canvassed its undoubted merits will be disappointed, but the decision of the majority on this issue seems quite clear. On the other hand, the Articles of Association, which govern the organization and running of the Australian Medical Association, including such controversial matters as the Federal Assembly, can be amended at a general meeting of the Association; so that if a majority decide after fair trial that, for example, deciding to have a Federal Assembly was a big mistake, appropriate changes can be made. The second thought, which is put forward for the benefit of those to whom it may appeal, is that the Parent Body has forecast a major increase in membership subscription rate from the beginning of 1962. If the Australian Medical Association is not in being by then, a very substantial additional sum will be required from members for transmission overseas. Some at least will think the point important. What matters, however, is that we should make up our minds in the adult fashion that is now appropriate and, with the added strength of a national medical association, set ourselves as a responsible profession to make the maximum contribution to the life and welfare of our rapidly growing nation.

The one comment that remains to be made is that our profession is deeply in debt to the members of the steering committee—the President of the Federal Council, Dr. H. C. Colville, Dr. A. J. Murray, Dr. W. F. Simmons, Dr. R. H. Macdonald, Dr. E. F. Thomson, and the General Secretary, Dr. J. G. Hunter. Their herculean task has demanded very many hours and a great deal of thought from men whose professional life already fully occupies both time and mind. Yet they have done what was asked of them well and willingly in a sense of service. It is important that it should be recognized.

Current Comment.

CHANNEL SWIMMERS.

Australian visitors to Britain are apt to find the sea temperatures around its shores uninviting, even at the height of the English summer. Nevertheless, ever since Captain Matthew Webb's pioneering feat in swimming unaided from France to England in 1875, the English Channel has appeared as an invitation and a challenge to long-distance swimmers from many parts of the world. Since Webb's day, more efficient methods of swimming, a better understanding of tides and currents based on past experience, as well as other refinements have combined to make the undertaking less formidable, if still arduous, and nowadays scarcely a year passes without someone adding his or her name to the list of successful contestants. In some years races are held, with a dozen or more participants, several of whom may achieve the crossing.

From the medical point of view, the interest of this sport lies in the problem as to how Channel swimmers are able to swim for 12 to 22 hours in water at a temperature of about 60° F. (15·5° C.) when after shipwreck persons survive in water at this temperature usually for only four to six hours. The study of this question was taken up by L. G. C. Pugh and O. G. Edholm of the Division of Human Physiology, National Institute for Medical Research, London, and a first account of their investigations was published some years ago;[1] they have now published the results of a more detailed study,[2] undertaken in collaboration with several other investigators, of the swimmers in the 1955 cross-Channel race. With the assistance of the Royal Navy, it was possible to follow the race and to examine swimmers who had to give up within a short time of their being taken out of the water. In this way 15 male and four female swimmers were examined prior to and during the 1955 cross-Channel race.

The somatotype of each subject was determined, and the swimmers were found to have high ratings for the mesomorphic and endomorphic components in the Sheldon classification. Skin-fold thickness, total body fat, and energy expenditure were measured. All the swimmers had thicker skinfolds, indicating more subcutaneous fat than is found in the average individual, and Pugh and Edholm state that when energy expenditure is high, an extra 1 mm. in the thickness of subcutaneous fat may be equivalent to raising the temperature of the water in which the swimmer is immersed by as much as 1·5° C. They conclude that the swimmer's greater thickness of subcutaneous fat probably plays an important role in preventing the development of hypothermia. This is illustrated by the fact that one of the fastest but leanest swimmers had a rectal temperature well below normal on completing the race, whereas slower fatter swimmers had rectal temperatures in the normal range. The relatively thin layer of lanolin or petroleum jelly that some swimmers apply to their bodies is for the same reason thought to be of value in conserving body heat. However, in their earlier communication Pugh and Edholm note that there appears to be some loss of confidence among Channel swimmers in the effectiveness of grease, and quote one woman swimmer, who was asked why she did not use grease, as saying that she could keep warm without it and saw no point in carrying the extra weight across the Channel (disregarding, of course, the fact that fat is buoyant in water). Three methods were employed to estimate the total of body fat—from total body water by the tritium dilution method, from total body water by the urea method of McCance and Widdowson, and by the density method of Behnke *et alii* (the subject being weighed in air and water); there was fair agreement between the results of these three methods. It transpired that though the swimmers had more subcutaneous fat than the average, the total body fat was below average, probably because of proportionately greater muscle development. Also, the fastest swimmers were those with least body fat.

It was noted that all the competitors observed used the crawl stroke, and maintained a virtually constant stroke rate until the last few hours, when signs of fatigue set in and the rate declined. Studies during practice before the race had shown a good correlation between speed and stroke rate, and this was held to justify the calculation of changes in speed during the race from alterations in stroke rate. The stroke rates observed during the race were comparable with those during practice, except in the later stages when the swimmers began to tire. It was not possible to measure oxygen consumption during the race, but such measurements were made during practice swims, and from these and from time and motion studies during the race, it was calculated that the energy expenditure involved in swimming the Channel is of the order of 10,000 Calories. Food intake during the race was relatively small, and Pugh and Edholm consider that it probably did not exceed 1000 to 1500 Calories. They state that one reason for the low food intake is that the swim-

[1] *Lancet*, 1955, 2: 761 (October 8).
[2] *Clin. Sci.*, 1960, 19: 257 (Number 2).

mers are not permitted to touch the side of the boat and do not stop for more than a minute at a time for fear of cramp. They note that in recent years the value of glucose and more frequent feeds has been recognized, and that most swimmers now take milk in one form or another with added glucose or sugar. They conclude that the greater part of the energy expenditure is probably derived from fat metabolism.

In their earlier paper, Pugh and Edholm mention some additional effects of hypothermia and discuss the practical application of their findings. In the 1954 race, when the water was somewhat colder (61° F.) than in 1955 (64·4° F.), two swimmers on leaving the water had rectal temperatures of 94·5° F. and 94·6° F. respectively. This raised the question of whether trained Channel swimmers tolerate hypothermia better than others, but the evidence on this point is inconclusive. On another point Pugh and Edholm appear to arrive at opposite conclusions in their two papers. In their earlier paper they gave the results of the measurement of heat loss from subjects lying still in a bath of cold water and from the same subjects when swimming in water at the same temperature, and found that there appeared to be little difference in the rate of body cooling in the two situations. They are therefore inclined to question the advice of E. M. Glaser (1950).[2]

> Fit men, who are in danger of immersion in cold water, might be advised to swim or struggle as hard as they can for as long as they can. If they try to preserve their strength by clinging to wreckage or floating on their life belts, they will die from cold.

and suggest that the traditional naval advice to cling to wreckage and not to waste energy by swimming is probably correct. However, in their latest paper, Pugh, Edholm and their colleagues state clearly that fat becomes an effective means of body insulation only at high rates of heat flow—that is, when there is a high rate of endogenous heat production, as in active swimming. Glaser has calculated that the heat production of a subject swimming actively may be ten times that in the resting state. The answer perhaps is that only a well-insulated individual has much chance of surviving prolonged immersion in cold water, whichever advice is followed, but that he has a better chance if he keeps swimming.

PULMONARY EMBOLISM.

ONE of the most dramatic modes of death, particularly of patients in hospital undergoing surgical or more often medical treatment, is that of pulmonary embolism. Despite the trend towards earlier ambulation, physiotherapy and other prophylactic measures, in a considerable number of hospital autopsies (8% in the Radcliffe Infirmary at Oxford) it is the cause of death.

Professor P. R. Allison and his colleagues, M. S. Dunnill and R. Marshall,[1] have carried out experiments on dogs in the Nuffield Department of Surgery at Oxford, in an attempt to answer questions such as these:

> (1) What is the day-to-day history of thrombus that forms in peripheral veins, becomes detached in the circulation and lodges in the lung?
>
> (2) Why does a patient die from a clot which often seems too small to affect a large enough area of the pulmonary vascular bed?
>
> (3) If a clot lodges in a pulmonary artery without causing death, is it completely absorbed, leaving a full-lumen vessel with smooth intima and normal circulation, or does the vessel recanalize inefficiently and incompletely, with diminished flow and diminished lung function?

Their interest in the problem was stimulated by two patients with subacute pulmonary emboli. In the former, lobectomy was successfully performed, with removal of a thrombosed pulmonary artery. However, the patient died seven months later after complete infarction of the left lung. The second patient recovered after bilateral pulmonary embolectomy.

The object of their experiments on dogs was to find out if a non-fatal pulmonary embolus was absorbed completely. They made large single radioopaque blood clots from the dog's own blood in eight animals. With the dog under anæsthesia, the clot was then placed in the inferior vena cava or jugular vein. The passage of the clot was followed by cine-radiography: it usually passed through the heart and into the pulmonary vessels within six seconds. More surprising, the clots caused no change in pulse, systemic blood pressure or electrocardiogram. Repeated radiographic study showed the clots to be absorbed very rapidly without any opacity in lungs such as might be due to an infarct or pleural effusion. Histological examinations at varying intervals showed that the artery had returned to normal in 28 days.

Under the conditions of the experiment then, the authors obtained answers to their first and third questions, but not to the second, which, from the patient's point of view, is the crucial one. The one practical suggestion they offer is that the problem of pulmonary embolus is not the prevention or treatment of the single massive embolus, but the counteracting of a continuous process. They advise "a further effort to produce lysis of clots reaching the lungs" while at the same time due attention is paid to the problems of stagnation and thrombosis in the deep veins of the legs.

It would seem that much more experimental work is required to answer the problem which has puzzled clinicians and pathologists alike: why should a patient collapse and die from an embolus not big enough to obstruct the pulmonary circulation as much as a ligature round one main branch, a common and safe surgical procedure nowadays?

HÆMORRHOIDS IN THE ADULT MALE.

IN a report of "a small epidemiological study" of hæmorrhoids in the adult male, R. M. Acheson and a post-graduate study group[1] point out that hæmorrhoids are widely agreed to be a common condition, yet there is not much satisfactory evidence as to how common it is, nor is a great deal known of the causes. Many theories have been advanced as to the cause, but few of these theories are supported by any facts. Thus Acheson and his colleagues have attempted to study the prevalence and ætiology of hæmorrhoids by comparing a group of 24 men who had undergone radical surgery with a control group of 26 men, chosen as a random sample from an electoral roll. These authors conclude, from their own findings and from a survey of the literature, that the prevalence of hæmorrhoids in adult males in England is in the order of 8% to 10%. Such a statement is open to criticism, since the absolute numbers in this series were very small—4 men in 30 stated that they suffered from hæmorrhoids. Further, the only evidence that these men did in fact have hæmorrhoids is their own statement. This is likely to be quite inaccurate, since, to most patients and even to a few doctors, any lesion around the anus, from pruritus ani to perianal hæmatoma, is "hæmorrhoids".

As regards possible ætiological factors, a number of conclusions stand out. The patients who had undergone surgery for their hæmorrhoids had a much higher incidence of a family history of this complaint and were predominantly members of the "upper" social classes (Registrar-General's Classes I and II). An association between hæmorrhoids and constipation was shown. Standing at work is considered a very likely causative factor in hæmorrhoids, since it was found that the work and recreation of the hæmorrhoid patients involved an excessive amount of standing. This could be the most significant finding in this study, since it points the way to future investigation and possibly eventual prophylaxis.

[2] *Nature (London)*, 1950; 166: 1068 (December 23).
[1] *Thorax*, 1960, 15: 273 (December).

[1] *Guy's Hosp. Rep.*, 1960, 109: 184.

Abstracts from Medical Literature.

HYGIENE.

Epidemiology of Childhood Accidents.

R. A. STALLONES AND L. CORSA (*Publ. Hlth Rep.* (*Wash.*), January, 1961) describe a study of accidents among children designed to develop a method of estimating the extent of accidents in a district, to gather information on which to base preventive programmes, to establish measurements for use in evaluating prevention programmes, and to provide background for more intensive study of specific accidents. By means of brief report cards completed by the casualty department staffs of hospitals in two Californian counties, the incidence of accidental injuries to children under 15 years of age was calculated as 78·2 per 1000 children per year. Reports from practising physicians indicate that an approximately equal number receive private treatment. The total incidence of medically attended accidents in these counties is therefore about 150 per 1000 children per year. More than half the cases reported from the hospitals were minor, and only 4% required in-patient care. The age at greatest risk of accidental injury was between two and three years; certain categories, however, showed an increasing risk with increasing age throughout the age span under study. The reported accident rates for boys and girls were 96 and 58 per 1000 children per year respectively. The rate for boys rose from a slightly increased rate in infancy to almost double the rate for girls among the 10 to 14 year old group. During June the daily accident incidence was approximately twice the rate during January. Poisoning accounted for 8% of the reported accidents. Of these 53% and 15% were by medicines and insecticides respectively. The highest incidence of accidental poisoning occurred in children aged from two to three years. Burns made up 3% of the total accidents reported. Of these, 36%, 7% and 57% resulted from hot liquids, fire or explosion, and from falling against hot objects, respectively. Of the 27,623 injuries reported, 1540 were due to automobile accidents and 1060 occurred in games and sports activities. Much of the information in this article is clearly shown in graphs and diagrams.

Epidemiology of Poisoning Accidents.

H. M. CANN *et alii* (*Amer. J. publ. Hlth*, December, 1960) present a study of approximately 15,000 reports of poisoning accidents forwarded to the United States Health Service between 1954 and 1958. About 90% of the incidents involved children under five years of age. Males predominated in the 5 to 9 years age group, were equally involved in the 10 to 14 years age group and were exceeded by females among those aged over 15 years. The period in which poisoning occurred most frequently was at the age of 18 to 24 months. Two-year-olds ingested internal and external medicines and cosmetics more frequently than did any other age group. Household cleaning and polishing agents, pesticides, petroleum products and paint and paint solvents were ingested most frequently by one-year-old children. Internal medicines comprised a higher proportion of substances ingested by three and four-year-old children than by other age groups. External medications and pesticides were ingested in highest proportion by one-year-olds and their relative importance declined with age, as did that of cleaning agents and petroleum products. Aspirin (largely "baby" type) accounted for half the internal medicine group and for 20% of all accidents. Poisonous or potentially poisonous substances were not in their original containers in one-third of all cases. Bottles were the most frequent substitute containers. The younger the child, the more frequently he ingested substances in substitute containers. Medicines and cosmetics were ingested proportionately more frequently in colder months, with petroleum products, paint solvents and pesticides more often in warmer months. The kitchen was the most frequent place of ingestion. In only 7% of cases was there no supervision by parents.

Screening Methods for Phenylketonuria.

W. R. CENTERWALL *et alii* (*Amer. J. publ. Hlth*, November, 1960) refer to recent knowledge that mental deficiency due to phenylketonuria is preventable if the disease is diagnosed in early infancy and proper dietary control instituted. They state that routine screening of all well babies is essential, and three screening programmes for the detection of phenylketonuria in infants are described. All mentally defective persons should also be screened and relatives of any found to have phenylketonuria informed so that infants in the family group can be examined and given the benefit of early treatment if found to be suffering from the disease. Six tests are outlined and the advantages of each discussed. The tests described are: (i) the test-tube test with ferric chloride, (ii) the diaper test with ferric chloride, (iii) the "Phenistix" (dip-stick) test, (iv) the filter paper test with ferric chloride, (v) the dinitrophenylhydrazine test-tube test, and (vi) the serum phenylalanine determination. The first five of these are simple urine tests. The second, third and fourth tests are particularly suitable for mass well-baby screening programmes. All the tests studied are comparable in accuracy and each test has enough specific merits to make it particularly valuable under certain circumstances. The maximum value of each type of test is dependent on some simple but specific precautions and knowledge essential to the reliability of the test. Most of these vital factors are included in the discussions of the respective tests.

The Bromination of Swimming Pools.

J. K. JOHANNESSON (*Amer. J. publ Hlth*, November, 1960) has studied the chemistry of bromamines and their bactericidal action in swimming-pool waters when bromine is used in water treatment. A break-point reaction occurs with ammoniacal nitrogen, but the decomposition of bromamines is more rapid than that of chloramines. There is also no bromine analogue of the irritating nitrogen trichloride. Also, the bactericidal properties of monobromamine are almost equal to those of free bromine. For this reason the author considers that it is not necessary to reach break point when bromine is used to purify water, and a small combined bromine residual is all that is necessary for effective purification. Details of the determination of free or combined bromine by the use of standard amperometric apparatus are given, but the author states that for practical purposes a direct reading on a chlorine comparator with either free or combined bromine will give bactericidal effects similar to the same concentration of free chlorine at the same pH.

Safety in the Public Health Laboratory.

E. B. M. COOK (*Publ. Hlth Rep.* (*Wash.*), January, 1961) outlines steps taken to prevent accidents and accidental infections in the laboratories of the Texas State Department of Health. A survey which revealed 20 laboratory infections among laboratory workers for the period 1930–1950 is summarized under headings showing type and year of infection, source of infection, confirmatory tests and comments. Thirteen different types of infection occurred. Other accidents included nine severe cuts, three broken bones from falls on stairways or uneven floors and six severe burns, one due to an ether explosion. A new laboratory occupied in 1958 contained safety features developed as a result of previous safety experience. Work with viruses and rickettsiæ is conducted in a separate building. Tuberculosis and mycological diagnostic work is done in an isolated area. Bacteriological safety cabinets are used for work with pathogens. Eye baths and overhead showers are available for splash accidents. Facilities are provided for control of infections in laboratory animals. Recent surveys revealed the existence of minor hazards such as door stops placed too far into passages, areas of concentrations of wax on floor surfaces and wet glazed tile floors. Safety procedures for routine work and for adoption in the event of accidents have been evolved and workers are required to follow them. Constant attention to the existence of laboratory hazards, orientation and education of new workers in laboratory safety procedures, continued emphasis on safety for old employees, and maintenance of protective immunization for "at-risk" workers, wherever possible, are the main measures stressed to reduce laboratory accidents and infections to a minimum.

Maximum Acceptable Concentrations.

H. B. ELKINS (*Arch. Environmental Hlth*, January, 1961) compares the widely recognized maximum allowable concentrations (M.A.C.) of toxic substances in occupational environments adopted by the American Conference of Governmental Industrial Hygienists with those accepted in Soviet Russia. In more than half of the irritant gases and vapours the values

are similar; others differ greatly. The Russian value for ethylene dioxide is 0·6 p.p.m., the American 50 p.p.m.; the Russian values for benzol, toluol and xylol are 6, 13 and 12 p.p.m. respectively compared with the American 25, 200 and 200 p.p.m. respectively. There are similar marked differences with the chlorinated hydrocarbons. The American M.A.C. for seven of these compounds is more than 15 times that permitted in Russia. The Russian values for carbon tetrachloride, trichloroethylene and ethylene dichloride are 3, 9 and 2·5 p.p.m. respectively, compared with American values of 25, 200 and 100 p.p.m. for the same substances. With the inorganic dusts and fumes the agreement between Russian and American values is, with a few exceptions, quite good. The exceptions include lead, mercury and manganese. In the author's opinion some of the Russian values (lead, mercury, carbon disulphide and carbon monoxide) are unreasonably low. On the other hand, it is probable that a number of the M.A.C.'s used in America are unrealistically high. In some cases, industrial exposures at the M.A.C. level have never occurred for other than brief periods. In others only a limited population has been exposed, for limited periods, to the M.A.C. In most cases where the American values exceed the Russian by more than a factor of 10 there is some evidence that the former figure is high. However, the author considers that in most such cases the Russian figure seems unreasonably low. Russian values are listed for a number of substances for which the Committee on Threshold Limits has made no recommendations.

PHYSIOLOGY.

Relation of Coronary Flow to Oxygen Supply.

A. GUZ *et alii* (*Amer. J. Physiol.*, July, 1960) state that coronary flow, heart rate, myocardial oxygen consumption and Walton strain gauge tension were determined in the isolated rabbit heart perfused with hæmoglobin solutions of varying oxygen content. Perfusion was carried out under constant pressure and with the hæmoglobin solution in equilibrium with 3% carbon dioxide and 97% air under atmospheric tension. Oxygen content was varied from 2 to 18 volumes per centum by diluting the hæmoglobin with Ringer-Locke solution. Change from a higher to lower oxyhæmoglobin concentration resulted in increased coronary flow; the reverse led to decreased flow. Heart rate, myocardial tension and oxygen consumption were constant at oxygen capacity above 2 volumes per centum.

Tactoid Formation in Deer Hæmoglobin.

J. H. MOON (*Amer. J. Physiol.*, July, 1960) reports that sickle cell preparations were made on eight Virginia white-tail deer killed during the 1958–59 hunting season. All preparations showed sickling. Paper electrophoresis of carboxyhæmoglobin from these animals revealed identical mobilities and a single hæmoglobin component present at pH 8·6. Alkali denaturation of the deer carboxyhæmoglobin solution showed that it was markedly resistant to alkali denaturation and that the kinetics of the denaturation were different from those of human cord hæmoglobin. Deer carboxyhæmoglobin migrated $0{\cdot}6 \times 10^{-5}$ cm.^{2}/volt/sec^{-1} faster than human carboxyhæmoglobin A in moving boundary electrophoresis in "Veronal" buffer at pH 8·2. Tactoids were demonstrated in free hæmoglobin solution with phase microscopy after concentration of the solution. These particles are very similar morphologically to those prepared from hæmoglobin solution containing hæmoglobin S.

Renal Tubular Reabsorption of Protein.

W. LATHEM AND B. R. DAVIS (*Amer. J. Physiol.*, October, 1960) report that by the use of the technique of stop flow analysis, renal tubular reabsorption of egg albumin and β-lactoglobulin was demonstrated in the dog. The stop flow patterns of these proteins were identical. Both egg albumin and β-lactoglobulin were shown to be reabsorbed in a proximal part of the nephron overlying the site in which PAH is secreted. Previous studies by stop flow analysis have demonstrated that hæmoglobin is reabsorbed in a similar proximal site. These experiments in the intact animal indicate, in agreement with histological studies, that renal tubular cells have the property of reabsorbing a variety of different types of proteins from glomerular filtrate, and that this property is apparently restricted to cells of the proximal convoluted tubule.

Afferent Initiation of Shivering.

C. M. BLATTEIS (*Amer. J. Physiol.*, October, 1960) reports that the activation of shivering was investigated in 30 lightly "Nembutalized" dogs by surface cooling of the left rear leg amputated at the hip, excepting the bone, the femoral and sciatic-peroneal nerves and the femoral artery and vein. The onset of shivering, although variable, was evidently not dependent on afferent impulses returning from the cooled leg because nerve section before or during cooling did not affect its occurrence; it appeared to be dependent on an effect of the cooled blood entering the truncal circulation because occluding the femoral vessels abolished shivering. There was not any apparent relation of the onset of shivering to deep body, mean skin and/or brain temperatures. The locus of action of the cooled blood in initiating shivering remained undetermined.

Breathing in Brief Exercise.

F. N. CRAIG AND E. G. CUMMINGS (*J. Appl. Physiol.*, July, 1960) report that two men ran for 20 or 60 seconds while inhaling air, oxygen or 4% carbon dioxide. Inspired respiratory minute volume was determined for each breath. Ventilation increased suddenly in the first breath with minimal changes in end-expiratory carbon dioxide tension and respiratory exchange ratio to a rate that remained constant for 20 seconds before increasing further. The rate of carbon dioxide output was uniform during the first 20 seconds. A 12% grade did not increase ventilation or oxygen uptake during runs of 20 seconds, but in the first minute of recovery ventilation was 64% greater than after level runs. Inhalation of oxygen inhibited ventilation by 24% in the 20-second periods before and after the end of a 60-second run. Inhalation of carbon dioxide begun at rest produced increments in ventilation and end-expiratory carbon dioxide tension that varied little during running and recovery. In the 20-second runs ventilation varied with speed but appeared independent of ultimate metabolic cost. The initial increase in ventilation was so rapid that there did not appear to be time for changes in chemical composition in the blood perfusing the muscles to reach the respiratory centre. For this reason it was necessary to postulate an as yet unidentified nervous mechanism. Thus, the initial rapid ventilatory rise and plateau offer an opportunity to study the nervous factors uncomplicated by the chemical. If the nervous factors drop out at the end of exercise as quickly as they enter at the beginning, the immediate recovery period should exhibit the chemical factors operating in a strength very little less than that effective during exercise.

Experimental Production of Intra-arterial and Intravenous Thrombi.

M. FRIEDMAN *et alii* (*Amer. J. Physiol.*, November, 1960) report that small segments of special alloy wire (98% magnesium and 2% aluminium) were pretreated by immersion in 0·5% zinc chloride for 15 to 25 minutes. Such wires disintegrate by focal electrolysis when inserted in the aorta or inferior vena cava of rabbits or rats and in so doing cause the formation of relatively uniform white thrombi. In the artery of the rabbit such thrombi were quickly overgrown by intimal fibroblastic tissue which also penetrated each thrombus. In the artery of the rat, this reaction was much less luxuriant and slower than in the artery of the rabbit. It is emphasized that the late arterial sequel to this thrombus resembles a pearly plaque.

Blood Eosinophil Rhythm in the Hamster.

F. HALBERG AND A. P. CHAUDHRY (*Amer. J. Physiol.*, November, 1960) report that in the absence of stimulation other than daily routine, Syrian golden hamsters show 24-hour periodic changes in eosinophil count. Animals standardized on a regimen providing for light from 0600 to 1800 hours alternating with 12 hours' darkness, have high eosinophil levels around noon and lower ones around midnight. The timing of hamster eosinophil rhythm corresponds to that recorded earlier for other nocturnal mammals, being out of phase by about 12 hours with the timing of the same rhythm in diurnally active species. Physiological changes with periods of about 24 hours have been recorded for the hamsters' gross motor activity rectal temperature, heart rate, oxygen consumption and epithelial mitotic activity.

British Medical Association.

MEETING OF THE FEDERAL COUNCIL.

A MEETING of the Federal Council of the British Medical Association in Australia was held on March 17, 18 and 19, 1961, at B.M.A. House, 135 Macquarie Street, Sydney. The President, DR. H. C. COLVILLE, was in the chair.

REPRESENTATIVES.

The following representatives of the Branches were present:

New South Wales: Dr. W. F. Simmons, Dr. A. J. Murray, Dr. R. H. Macdonald, Dr. E. F. Thomson.

Queensland: Dr. R. A. M. Miller, Dr. Charles Roe.

South Australia: Dr. L. R. Mallen, Dr. C. O. F. Rieger.

Tasmania: Dr. L. N. Gollan, Dr. F. R. Fay.

Victoria: Dr. H. C. Colville, Dr. J. G. Johnson, Dr. T. G. Swinburne.

Western Australia: Dr. C. W. Anderson, Dr. D. M. Clement.

APPOINTMENT OF OFFICE BEARERS.

The following office bearers for the ensuing year were elected unopposed: *President*, Dr. H. C. Colville; *Vice-President*, Dr. A. J. Murray; *Honorary Treasurer*, Dr. W. F. Simmons.

ANNUAL REPORT.

The General Secretary presented the Annual Report for the year 1960. This was adopted.

FINANCE.

Annual Financial Statement.

The Honorary Treasurer, Dr. W. F. Simmons, presented the annual financial statement. He pointed out that in estimating the expenses for 1961 special note had been made of the extra costs that would be associated with the formation of the new medical association, and in particular provision had been made for four meetings of the Federal Council should they be necessary. The financial statement was adopted.

Organization Fund.

The Honorary Treasurer presented the financial statement of the organization fund for the year ended December 31, 1960. The balance of £1703 at the end of 1959 had been reduced to £1473 because part of the expenses of the Federal Council meeting held in conjunction with the convention to discuss the new medical association had been borne by the fund.

Independence Fund.

The Honorary Treasurer presented the financial statement of the federal independence fund for the year ended December 31, 1960. The credit balance of the fund was £26,046.

Entertainment Fund.

The Honorary Treasurer presented the financial statement of the entertainment fund for the year ended December 31, 1960. The balance of £279 at which the fund had stood at the end of 1959 had been reduced to £33 15s., mainly as the result of the entertainment of delegates to the convention to discuss the new medical association.

Auditors.

It was resolved that Messrs. F. W. Duesbury and Co. should be reappointed auditors for the ensuing year.

HENRY SIMPSON NEWLAND PRIZE IN SURGERY.

The Honorary Treasurer presented the financial statement of the Henry Simpson Newland Prize Fund for the year ended December 31, 1960. The credit balance of the fund stood at £1104. It was emphasized in discussion that there was need for publicity in relation to the prize as the number of entries received in the past had been disappointing.

MEDICAL OFFICERS RELIEF FUND (FEDERAL).

On behalf of the trustees, Dr. W. F. Simmons presented the report of the Medical Officers Relief Fund (Federal) for the year ended December 31, 1960. This showed that the total assets of the fund stood at £6329. During the year an amount of £364 had been paid to three beneficiaries.

FEDERAL MEDICAL WAR RELIEF FUND.

On behalf of the trustees, Dr. W. F. Simmons presented the trustees' report of the Federal Medical War Relief Fund for the year ended December 31, 1960. The total assets of this fund stood at £16,214. During the year an amount of £854 had been paid to nine beneficiaries.

HONOURS.

The General Secretary reported that letters of congratulation had been forwarded to the following members of the medical profession who had received honours in the New Year Honours List: Sir Rowden White, Kt., C.M.G.; Professor Sir Brian Windeyer, Kt.; Professor Sydney Sunderland, C.M.G.; Dr. E. H. Derrick, C.B.E.; Dr. Una Porter, O.B.E.; Dr. D. E. Trumpy, M.B.E. A letter of congratulation had also been sent to Major A. F. Seymour, M.B.E., of the Royal Australian Army Medical Corps. The General Secretary referred also to the award of the Nobel Prize in Medicine for 1960 to Sir Macfarlane Burnet and reported that a letter of congratulation had been sent to him.

GOLD MEDAL OF THE BRITISH MEDICAL ASSOCIATION IN AUSTRALIA.

The Federal Council decided unanimously that the Gold Medal of the British Medical Association in Australia should be awarded to Dr. Henry Cecil Colville and to Dr. William Foster Simmons for distinguished service to the British Medical Association and to members of the medical profession in Australia. This decision was conveyed to Dr. Colville and Dr. Simmons by the Vice-President, Dr. A. J. Murray, during the meeting.

MEDICAL PRACTICE IN AUSTRALIA.

The General Secretary reported that he had had inquiries from over 40 doctors overseas requesting information on medical practice in Australia. In each case a suitable reply had been sent.

TWELFTH QUADRENNIAL CONGRESS OF THE INTERNATIONAL COUNCIL OF NURSES.

The General Secretary reported that the Victorian Branch of the British Medical Association was arranging an exhibition on behalf of the Federal Council at the Twelfth Quadrennial Congress of the International Council of Nurses which was to be held at Melbourne in April, 1961.

CONFEDERATION OF MEDICAL ASSOCIATIONS OF ASIA AND OCEANIA: SECOND GENERAL ASSEMBLY.

The General Secretary reported that Dr. K. C. Crafter of South Australia had been nominated as a Federal Council delegate at the Second General Assembly of the Federation of Medical Associations of Asia and Oceania to be held in Manila from April 22 to 30, 1961. A copy had been received of the proceedings of the first congress of the Federation which had been held in Tokyo in April, 1959.

ASIAN GASTRO-ENTEROLOGY SOCIETY.

The General Secretary reported that Dr. Geoffrey Hagarty of Sydney had been appointed the Federal Council delegate to a meeting to be held at Tokyo from March 30 to April 5, 1961, to discuss the formation of an Asian Gastro-Enterology Society.

INTERNATIONAL SOCIETY OF GEOGRAPHICAL PATHOLOGY.

The General Secretary reported that a letter had been received from Professor E. J. ten Seldam of the University of Western Australia, conveying a request from Professor E. Roulet of Switzerland for the establishment of a section of the International Society of Geographical Pathology in Australia. The matter had been referred to the appropriate colleges and was still under discussion.

SERVICES CANTEEN TRUST FUND SCHOLARSHIP.

The General Secretary said that he had received from the Services Canteen Trust Fund details of scholarships to be awarded for 1961 for post-graduate studies in auxiliary services to medicine, welfare and science. This had been passed on to the Branches for appropriate publicity.

LIFE ASSURANCE: FEE FOR EXAMINATION AND COMPLETION OF FORMS.

A letter was received from the New South Wales Branch recommending that a request be made to the Life Offices'

Association of Australasia to increase the fee for life assurance examination and completion of necessary forms from £2 2s. to £3 3s. It was pointed out that the current fee of £2 2s. had remained unchanged since July 1, 1953. The Federal Council approved this request. It also resolved to request that the fee payable to a general practitioner for submitting a report on a patient under his care be increased from £1 1s. to £1 11s. 6d.

Mailing of Restricted, Dangerous or Specified Drugs.

A further discussion took place on the mailing of restricted, dangerous or specified drugs in the light of a request from the President of the Australian Association of Ethical Pharmaceutical Industry for modification of postal rule 30. The request had been passed on to the Federal Council by the Director-General of the Postmaster-General's department. After consideration of the views of the Branches the Federal Council resolved that approval should be given to proposals made by the Director-General of the Postmaster-General's department that Restricted Drug and Specified Drug be removed from the exemption in postal rule 30 (5) (*b*) and included in the general text of the paragraph and be subject to the same conditions as poisons, and that other types of packing as approved by that department be permitted in all cases, instead of being restricted to poisons and narcotic drugs in tablet form as at present. It was considered that this would simplify the matter of postage and still be satisfactory.

Australian National Committee for World Refugee Year.

The General Secretary reported that the Assistant General Secretary, Dr. C. J. Ross-Smith, had represented the President, Dr. H. C. Colville, at the general meeting of the Australian National Committee for World Refugee Year in September, 1960. Dr. Ross-Smith presented a report of the meeting, at which it had been announced that the target amount set up by the committee in its appeal had been exceeded.

Matrimonial Causes Act.

At the meeting of the Federal Council in August, 1960, approval was given of the action of the President in advising the Attorney-General that the Federal Council considered that no further steps should be taken by Parliament to reintroduce a section in the *Matrimonial Causes Act* which provided that a marriage was voidable where, at the time of the marriage, either party to the marriage was subject to recurrent attacks of insanity or epilepsy. The General-Secretary reported that no reply had been received from the Attorney-General, but that the section concerned had not been included in the Act.

Artificial Respiration in Handbook of First Aid.

A letter was received from the South Australian Branch requesting that Federal Council draw the attention of the publishers of a handbook of first aid which had been issued as a supplement to the *Reader's Digest* of January, 1961, to an omission in the article, as no reference had been made to first attempting to drain the water from a person who was supposed to be drowned. It was reported that the matter was still under consideration.

Forensic Pathology Services in Australia.

A copy of a booklet was received from the College of Pathologists of Australasia containing recommendations with respect to forensic pathology services in Australia.

Australian Medical Congress.

Henry Simpson Newland Oration.

The Federal Council resolved to invite Dr. S. Gilder, Editor of the *World Medical Journal*, to deliver the Henry Simpson Newland Oration at the Australasian Medical Congress to be held in Adelaide in 1962.

Medico-Legal Section.

A recommendation was received from the New South Wales Branch that Federal Council consider a proposal made by the New South Wales Medical Defence Union, that steps be taken to form a medico-legal section at the next congress, but the idea was not approved.

Australasian Medical Publishing Company Limited.

Per-Capita Payment for The Medical Journal of Australia.

A letter was received from the Chairman of Directors of the Australasian Medical Publishing Company Limited, Sir Henry Newland, advising that there would be no alteration in the present per-capita payment for The Medical Journal of Australia. It would remain at 5s. for the year 1961, the receipts to be applied towards the purchase of Series E 2 debentures.

Australian Medical Association.

Formation.

At the meeting of the Federal Council in November, 1960, which immediately followed the convention to consider a draft constitution for the proposed Australian Medical Association, it was decided to ask the steering committee to consider the recommendations of the convention and to prepare subsequently a new draft of the constitution for consideration by the Federal Council. It had been prepared after consultation with the Council's legal adviser and was in the form of a draft of the Memorandum and Articles of Association. This draft was approved by the Federal Council and considered at length, and after the making of certain minor amendments was approved for final submission to the Branches. At the same time the Federal Council recorded its appreciation of the work of the members of the steering committee, the secretariat and the Council's legal adviser, Mr. J. O. Maddox, in the preparation of the draft.

Appointment of First Officers.

In order to complete the memorandum and articles of association of the Australian Medical Association it was necessary to nominate the first officers of the association. For this purpose the Federal Council decided to recommend to the Branches that the present Federal Council and office bearers be the interim Federal Council and office bearers of the Australian Medical Association until such time as the new council and office bearers could be elected in accordance with the constitution of the Australian Medical Association. As the office of chairman of the Federal Assembly had no counterpart on the present Federal Council, it was decided to recommend that that office on the interim council be filled by Dr. L. R. Mallen.

Provisions for Discipline of Members.

At the request of the Tasmanian Branch the question was considered of the provision in the constitution of the Australian Medical Association of means whereby effective discipline of members might be possible. The difficulties and desirable and undesirable aspects of this were discussed, but no new provisions were suggested that were likely to be generally acceptable.

Voting by Branch Representatives at Meetings of Federal Council.

The Western Australian Branch, in a letter, drew attention to the following statement from the 1960 B.M.A. Year Book relating to representatives of constituents in Great Britain:

> It should be appreciated that Representatives of Constituencies are not delegates from local units who are absolutely bound by instructions of the Constituency they represent. In speaking and voting at Representative Meetings, they must, of course, have regard to the instructions or opinions of their Constituents upon the matters to be discussed at Representative Meetings and so far as is possible they are expected to conform to the preponderance of opinion of the members they represent. But in a democratic body such as the Association the Representatives must of necessity be allowed reasonable discretion, and, if in the course of a debate in a Representative Meeting an issue is raised which was not considered at a local meeting, where a new aspect of a particular problem emerges from the discussion, or new information becomes available or new arguments are advanced, the Representative of the Constituency would be justified in exercising his discretion in voting if he felt that his decision would be endorsed by the members of his Constituency.

The Western Australian Branch Council expressed the opinion that it was most desirable that representatives attending Association meetings should be in a position to exercise discretion in voting as was provided in the United Kingdom. The question was raised whether that was the case with representatives of the Branches at Federal Council meetings, and it was asked that the matter be considered by the Federal Council. The General Secretary reported that the letter from the Western Australian Branch had been referred to the other Branches. The Queensland Branch Council, in a resolution, expressed its belief that its Federal

Councillors had served it with integrity and discretion and said that it would continue to rely on them to serve it in the presentation of its views. The New South Wales Branch Council said that its representatives were expected to conform to the opinion of the members that they represented, but that, if in the course of a debate in a Federal Council meeting an issue was raised which was not considered by the Branch Council or a new aspect of a particular problem arose in the discussion or new information became available or new arguments were advanced, the representatives would be justified in exercising their discretion if they felt that their decision could be endorsed by the Branch Council. The Victorian Branch Council said that its attitude had always been expressed in the passage from the B.M.A. Year Book quoted by the Western Australian Branch, and it supported the opinion of that Branch that representatives attending Federal Council meetings should be in a position to exercise discretion in voting as was provided in the United Kingdom. The South Australian Branch Council agreed with the views of the Western Australian Branch and stated that its delegates had been given discretionary powers similar to those referred to in the B.M.A. Year Book. The Tasmanian Branch Council was in favour of the recommendation of the Western Australian Branch. In view of this general agreement it was not considered that further action was necessary.

STATE ORGANIZATION COMMITTEES.

At its meeting in August, 1960, the Federal Council expressed the opinion that it was advisable to organize the medical profession forthwith in order (i) to review and improve the present national health scheme and (ii) to resist the growing practice of government and other instrumentalities of introducing sudden alterations of conditions of service without previous consultation with the profession through its organizations. To that end the Federal Council recommended that active organizational committees be set up in each State for the sole purpose of achieving that objective, it being pointed out that the activities of such committees must be coordinated at federal level. Reports were received from the Branches of activity in this direction, and in particular the representatives of the Victorian and Queensland Branches stated that the organization in their States was such that every member of the Branch could be communicated with at very short notice.

NATIONAL HEALTH SERVICE.

Further Extension of the National Health Service.

A letter was received from the New South Wales Branch referring to the attitude of the profession towards further extension of the National Health Service. This had been circulated to the other Branches, and the Federal Council discussed this subject in the light of the views expressed by the several Branches. It was appreciated that the matter had already been the subject of a great deal of previous discussion, and Federal Council had already made its views clear. However, it was decided to reaffirm the principles that in any future amendments or extensions of the *National Health Act* in which the medical profession was directly or indirectly concerned, unless it had been consulted and fully informed prior to their implementation the Government could not necessarily expect the profession to cooperate.

Pensioner Medical Service.

At its meeting in August, 1960, the Federal Council resolved to make a further approach to the Minister about an increase in the fees payable to medical practitioners under the Pensioner Medical Service. The Minister subsequently had stated that in his view no additional information had been brought forward to justify a further approach to Cabinet. As a result of further discussion at the meeting of the Federal Council in November, 1960, the Minister was again approached and the Federal Council's attitude was restated. At the same time it was suggested to the Minister that with the lapse of the agreement between the Government and the Federal Council on June 30, 1960, there no longer existed a form of agreement to provide general medical practitioner service for pensioners and their dependants, a situation which the Federal Council considered should be rectified. In his reply the Minister had restated his willingness to make a further approach to Cabinet, should Federal Council submit fresh evidence in support of increased fees. In addition he had pointed out that so far as the legislation governing the service was concerned, the terms and conditions of the agreement continued to operate without alteration until such time as a new agreement was made. The present arrangements would continue until it was mutually agreed to change them.

The Federal Council accepted the fact that in the terms of the *National Health Act* the arrangements which the Government had made with individual practitioners had remained in force until either party gave notice of intention to terminate the agreement, despite the lapse of the Government-B.M.A. agreement. However, with regard to the other aspect of the service under consideration it was decided to make a request to have the fees increased from 11s. to 13s. for surgery consultation and from 13s. to 15s. for domiciliary visit and for this purpose to arrange a conference between the Prime Minister and the Federal Council. It was agreed that at such a conference the Federal Council would be represented by the executive officers with Dr. J. G. Johnson and Dr. L. R. Mallen.

Participation of Specialists.

At its meeting in August, 1960, the Federal Council resolved to protest against the action of the Commonwealth Department of Health in extending the scope of the Pensioner Medical Service to include treatment by specialists without reference to the Federal Council. In a subsequent letter to the Minister it was pointed out that the Federal Council regarded the Pensioner Medical Service as essentially a general practitioner service, and reference was made to a statement by the Minister in a letter in 1958 to the effect that the medical service within the scope of the Pensioner Medical Service should be all such services as were ordinarily rendered by the general practitioner in the surgery or at the home, including the treatment of a patient who had undergone a surgical operation from the time that he had returned to his home from hospital. In his reply to the recent approach the Minister had stated that the paragraph quoted from his letter merely defined the scope and the nature of medical services covered by the Pensioner Medical Service. It did not define the status of the medical practitioners who might be enrolled in the service, nor did it restrict participation to general practitioners. If a specialist was prepared to provide those medical services in accordance with the terms and conditions of the agreement, he might be enrolled as a participating doctor. That policy had been consistently followed since the inception of the service, and the enrolment of specialists who were willing to provide general practitioners' services for pensioners under the agreement did not therefore represent a new agreement. Subsequently the New South Wales Branch recommended that an approach be made to the Royal Australasian Colleges and other specialist bodies to seek their opinion in the matter. The Federal Council, after discussion, reaffirmed its opinion that the present Pensioner Medical Service was a general practitioner service and should be restricted to members of the profession practising as general practitioners and decided to take the matter again to the Minister for Health.

Liberalization of the Means Test.

A letter was received from the South Australian Branch advising of concern in that State in regard to the increase in the number of persons with Pensioner Medical Service entitlement cards reporting to private practitioners, and asking that inquiries be made whether there had been an increase in the number of persons receiving these concessional services beyond 1954 level. The General Secretary reported that he had discussed the matter with the Department of Social Services and had been assured that the Pensioner Medical Service would not be liberalized.

Pensioners in "Public Beds".

A letter was received from the Victorian Branch advising of a satisfactory solution to a problem which the Branch had previously raised relating to pensioners hospitalized in benevolent homes classified as hospitals for the purpose of receiving the Commonwealth subsidy of 8s. per day, visiting medical officers being unable to claim fees for service. It appeared that no similar problem existed in South Australia, Western Australia, Queensland or Tasmania. The New South Wales Branch had advised that in that State there were a certain number of old people's homes which were classified as public hospitals, and in such cases the doctor was unable to make a charge for attendance on any pensioner.

Pharmaceutical Benefits.

At its meeting in August, 1960, the Federal Council set up a committee to examine alternative pharmaceutical benefits schemes and possible modifications of the present scheme for the purpose of making recommendations to the Federal Council. The committee consisted of Dr. W. F. Simmons and Dr. J. G. Johnson with power to coopt. The report was submitted to the meeting of the Federal Council

in November, 1960, and subsequently referred to the Branches for their comments. In a further consideration of the matter the Federal Council discussed a recommendation of the New South Wales Branch that the medical practitioner should be left entirely out of the transaction for the provision of pharmaceutical benefits; he should be able to write whatever prescriptions he thought necessary, and consideration of whether or not they were treated as pharmaceutical benefits should be a matter entirely between the pharmacist and the Government. In the discussion on this some comparison was made of the scheme with that prevailing in New Zealand. It was pointed out by Dr. L. R. Mallen that in practice the doctor was free to follow the course suggested at present, but his patients would be very likely to object. Dr. J. G. Johnson said that unless the practitioner who wrote the prescription took an interest in the scheme, he would from time to time deprive his patients of benefits to which they would be entitled if the provisions of the scheme were taken into account. He said that not a great deal of time was involved now that doctors had become familiar with the scheme. Dr. A. J. Murray expressed the view that the scheme should be designed to fit in with the doctor's habit of prescribing. Dr. T. G. Swinburne said that if the profession asked to be left right out of the operation of the scheme it might be regarded as having forfeited its claim to be consulted in any further developments of the scheme. Representatives of the Queensland and Tasmanian Branches said that the view of their Branch Councils was that the patient's welfare was what mattered. Dr. R. H. Macdonald said that it was not the doctor's role either to provide benefits or to deprive patients of benefits; he should be able to prescribe what he wished in any quantity he wished.

The Federal Council resolved not to adopt the suggestion of the New South Wales Branch, and it approved and adopted the Committee's report. After further consideration of the recommendations of the report it was decided to recommend to the Minister for Health that the list of restricted pharmaceutical benefits be abolished, and that if the Government should be unwilling to abolish the restricted list it should be urged that the list in any case be reviewed with the aim of drastically reducing the number of restricted drugs. The preparation of a list of the drugs which should be removed from the restricted list in the latter eventuality was left in the hands of the committee which had prepared the report.

On the question of limitations of maximum quantities the General Secretary reported that the Minister had advised that the matter was being negotiated with the chemists. To bring it into effect it would first be necessary to have a new agreement with them. The Minister's attitude was sympathetic.

Supply of Pharmaceutical Benefits to Inmates of Children's Homes.

At its meeting in August, 1960, the Federal Council decided to recommend to the Minister for Health that inmates of children's homes and similar benevolent institutions be exempt from the 5s. payment for drugs. The Minister in his reply had said that the Government in framing the revised pharmaceutical benefits scheme had given consideration to the class or classes of persons who should be exempted from paying the 5s. prescription fee, but after considering all factors had decided that only those who had entitlement cards under the pensioner medical scheme should be accepted. In the circumstances he regretted that he was unable to meet the wishes of the Federal Council in the matter.

In the light of a further letter from the New South Wales Branch referring to the financial difficulties that this had raised in a Salvation Army boys' home in Sydney, the Federal Council resolved to make a further approach to the Minister for Health in the matter.

Preparations for Immunization and Vaccination.

At its meeting in August, 1960, the Federal Council supported the recommendation of the Australian College of General Practitioners concerning the free issue of preparations for immunization and vaccination in such conditions as diphtheria, pertussis, poliomyelitis, smallpox, tetanus and typhoid fever, and subsequently referred the recommendations to the Minister for Health. In his reply the Minister mentioned the provision for the supply of poliomyelitis vaccine and quadruple vaccine, and also expressed his willingness to arrange for the supply of smallpox vaccine for any vaccination campaign arranged by a local authority or for a national campaign. After further discussions the Federal Council decided to take no further action in the matter.

Government Prescription Forms.

At its meeting in August, 1960, the Federal Council decided to request the Department of Health to discontinue the printing and distribution of prescription forms to private practitioners. The recommendation was subsequently conveyed to the Minister, who had agreed to it, distribution to cease when present stocks were expended.

Dispensing Fees Payable to Members Who do Their Own Dispensing.

Letters were received from the Victorian Branch and South Australian Branch relating to members who did their own dispensing. It was pointed out that an anomalous situation existed whereby chemists were paid dispensing fees for dispensing ready prepared preparations under the National Health Service while "approved doctors" were not. The General Secretary reported that the matter had been taken up with the Minister for Health, who had advised that in the Government's opinion the supply of drugs by approved doctors to patients was complementary to other professional responsibilities and the 33½% mark up on ready prepared drugs was sufficient to cover the service given. Doctors did not have to carry the same stocks as were required of pharmacists, nor did they have the same business risks. The Federal Council decided to make further inquiries and to consider the matter at the next meeting.

Pharmaceutical Benefits Advisory Committee.

At its meeting in November, 1960, the Federal Council approved in principle of the proposal of the Director-General of Health to enlarge the Pharmaceutical Benefits Advisory Committee by two additional members, such members to be appointed by the Minister for Health from a panel of four nominated by the Federal Council. As a result of further correspondence with the Director-General of Health the Federal Council decided upon a panel of four names to be submitted to the Minister for Health should it be requested to do so.

Statistics Relating to Pharmaceutical Benefits.

A letter was received from the New South Wales Branch requesting information on the percentage of prescriptions written for general pharmaceutical benefits, pensioner pharmaceutical benefits, and repatriation pharmaceutical benefits from the total number of prescriptions written. The General Secretary reported that he had discussed the matter with the Department of Health, but at present no reliable figures were available, particularly in relation to the total number of prescriptions written. The Federal Council decided to ask the Department of Health to furnish statistics relating to the number of prescriptions which were provided under the Pharmaceutical Benefits Scheme if and when they were available.

Authority for Doctors to Dispense Pharmaceutical Benefits.

A letter received from the Western Australian Branch requested that the subject of the authority for doctors to dispense pharmaceutical benefits be taken up with the Director-General of Health with a view to the delegation of authority to local Commonwealth Directors of Health to deal with applications. The General Secretary reported that advice had been received from the Director of Pharmaceutical Services in Canberra that the *National Health Act* provided the authority only to the Director-General of Health and the Director of Pharmaceutical Services to approve application. In discussion this opinion was questioned, and the Federal Council resolved to direct the attention of the Director-General of Health to the provisions of the *National Health Act* in the matter.

Medical Benefits.

Leaflets.

A letter was received from the Minister for Health drawing attention to the fact that doctors in one State were posting a leaflet with their patients' accounts suggesting that, if not already a contributor, the patient should join a medical benefits fund which was named. The Minister asked for the views of the Council in the matter. After considering views of the individual Branches the Federal Council decided to inform the Minister for Health that it was of the opinion that the distribution of leaflets advocating that the public join a medical benefits organization should not specify one

organization, but should list the names of all registered medical benefit organizations in the State concerned.

Anomalies in Schedule of Benefits.

A letter was received from the New South Wales Branch forwarding a list of anomalies in the schedule of benefits. The General Secretary reported that these had been referred to the Minister for Health, who had in general accepted the recommendations with a view to their incorporation in the schedules. Subsequently the Victorian and Tasmanian Branches had protested that the fee allowed for hypothermia amongst the new items was inadequate. After discussion the Federal Council decided to obtain the views of other Branches and the Australian Society of Anæsthetists on the matter.

Services Rendered on Behalf of Medical Practitioners.

The General Secretary reported that a letter had been received from the College of Pathologists of Australia forwarding a criticism of a statement in the monthly bulletin of the New South Wales Branch in May, 1960, relating to the referral of patients for pathological examinations and submitting further arguments against the referral of patients to technologists. The N.S.W. Branch, on being approached on this matter, advised that it was in full accord with the policy expressed by the College, and decided to draw its members' attention again through the Monthly Bulletin to the necessity to send patients to fully qualified practitioners and not to medically qualified technicians. The College had been advised of this answer.

Hospital Benefits.

Special Accounts.

A letter was received from the New South Wales Branch drawing attention to the difficulty experienced by persons over the age of 65 years, and those who had been transferred to the special accounts list, in obtaining hospital benefits, including hospital contribution fund benefits, when in-patients of smaller private hospitals. The matter had been referred to the Branches, and in the light of their views the Federal Council resolved to make representations to the Commonwealth Department of Health, so that persons over the age of 65 years, and those who had been transferred to the special accounts list, should be able to obtain hospital benefits to which they were legally entitled because of the acute nature of their disability when in-patients of smaller private hospitals.

Waiting Period.

A letter was received from the New South Wales Branch asking the Federal Council to take up with the Commonwealth Government the matter of the present waiting period of two months which must elapse prior to entitlement to hospital benefits, with a view to having this waiting period eliminated. The Federal Council discussed the matter and its practicability, but took no action.

Commonwealth Health Insurance Council.

A report was received from Dr. A. E. Lee, who had represented the Federal Council at the meeting of the Commonwealth Health Insurance Council in November, 1960. A copy of the report had been circulated.

Medical Services Committee of Inquiry.

Reference was made to notices in the *Commonwealth Government Gazette* of reprimand of three medical practitioners as the result of the investigations of Medical Services Committees of Inquiry.

Vaccination against Poliomyelitis.

A discussion took place on the question of the distribution and control of poliomyelitis vaccine and quadruple vaccine, and it was decided to approach the Department of Health with a request that steps be taken to simplify their distribution.

Descriptive Titles in Group Practices.

At the suggestion of the New South Wales Branch and with the concurrence of the other Branches the Federal Council decided to draw attention to its decision of August, 1954, which was in the following terms:

> That Federal Council is not opposed to the use of the term "clinic", but is opposed to the title of newly-formed groups being associated with the name of a suburb, town or district.

General Practitioner Training at Undergraduate Level.

A letter was received from the Australian College of General Practitioners requesting stronger support of a plan to have Dr. Robert Morrison of the Rockefeller Foundation review general practitioner training in Australia. Federal Council, having previously decided to support this plan, reiterated its views.

British Medical Association.

Annual General Meeting, Auckland, February 4 to 10, 1961.

The General Secretary referred to the annual general meeting of the Federal Council of the British Medical Association which had been held in Auckland from February 4 to 10, 1961, in conjunction with the biennial conference of the New Zealand Branch. He and the Assistant General Secretary, Dr. C. J. Ross-Smith, had attended. After the meeting a party of officers and members of the Parent Body had visited Australia and some members of the party had visited each State. The interstate air travel costs were paid by the Federal Council.

After discussion the Federal Council decided that the accommodation costs of the overseas visitors should be shared amongst the Branches on a membership basis. The Federal Council also resolved that each Branch should be thanked for its cooperation in entertaining the Federal Council's guests, the officials of the Parent Association, during their visit. A letter of thanks from Dr. S. Wand, Chairman of the Council of the Parent Body, to the President of the Federal Council, Dr. H. C. Colville, was published in the Journal in the issue of March 25, 1961.

Presentation of Gavel and Block.

It was reported that the Parent Body had prepared a gavel and block for presentation to the Australian Medical Association. It was understood that this would be officially presented at the inaugural meeting of the new association in 1962.

Roll of Fellows.

Advice was received from the Queensland Branch that Dr. Harold Horn, Dr. Alan E. Lee and Dr. J. G. Wagner had been elected Fellows of the British Medical Association. The Tasmanian Branch had advised that recommendation for admission to the Role of Fellows was being made for five of its members.

Sixth British Commonwealth Medical Conference.

Dr. T. G. Swinburne presented a report of the sixth British Commonwealth Medical Conference held in Auckland from February 1 to 4, 1961. Dr. Swinburne had attended the conference as the delegate of the Federal Council with Dr. H. C. Colville as assistant delegate. The Federal Council expressed its thanks to Dr. Swinburne for his very able representation at the conference and for his excellent report.

Canadian Medical Association Delegation.

The General Secretary reported that a visit to Australia had been made between February 8 and March 4, 1961, by a delegation from the Canadian Medical Association comprising Dr. and Mrs. T. J. Quintin, Mr. and Mrs. B. E. Freamo, Dr. T. C. McCoy and Dr. Peter Banks. The purpose of the visit had been to ascertain at first hand details of the operation of the Australian health insurance schemes.

National Health and Medical Research Council.

Copies were received of the official report of the forty-ninth session and a personal report by Dr. W. F. Simmons on the fiftieth session of the National Health and Medical Research Council, on which Dr. Simmons was the Federal Council representative. The Federal Council thanked Dr. Simmons for his excellent report.

National Advertising Code.

A copy of a draft of an Australian code of standards for the advertising of proprietary medicines was received from the Director-General of Health, copies of which had been sent direct to Branches for their comments. Particular reference was made to the question of radio talks by pharmacists. The Federal Council expressed the opinion that a pharmacist was not qualified to discuss publicly diseases and their treatment, and that any discussion on those subjects should emanate from a qualified medical

practitioner, preferably anonymously, and only under the ægis of a university, a learned society or college, a government depatment or a health education council, and with the permission of the British Medical Association.

COMMONWEALTH HEALTH DEPARTMENT.

Interim Report by the Director-General of Health.

An interim report for the period 1959-1960 was received from the Director-General of Health of the Commonwealth Health Department.

Sending Patients Abroad for Medical Treatment.

A letter was received from the Minister for Health in which he referred to the fact that the Commonwealth Government was from time to time under pressure to provide funds to assist Australians to go overseas for the purpose of obtaining treatment against some particular condition of which there was believed to be no knowledge or cure in Australia. In most cases the affected person had been sent as the result of local help and subscription, and it had been usual for the Commonwealth Government to decline to assist on the ground that it had no special funds available, and also had no jurisdiction in selecting the persons who might go overseas. The Minister said that it might be that occasionally there was a genuine case in which treatment was available overseas but not in Australia. In such circumstances it might seem that Government assistance would be warranted. The difficulty arose, however, in ascertaining which were genuine cases and which were not. The genuine cases would need to be those in which no treatment was available in Australia and in which a course of treatment recognized as having been successful was available overseas. The Minister asked Federal Council's views on the subject and in addition wished to know whether Federal Council would accept responsibility of selecting a board of specialists for each case should the Commonwealth Government decide to provide financial assistance. After discussion Federal Council expressed its willingness to appoint panels of specialists in particular cases where the Government required advice as to the necessity of sending patients abroad for treatment which was not available in Australia.

Visitors from Canada.

The General Secretary reported that correspondence had been carried on with the Director-General of Health regarding the visits (i) of Dr. K. C. Charren, Director of Health Services of Canada, and (ii) of three members of the Governmental Advisory Committee on Medical Care of Saskatchewan Province in Canada, and that at the Director's request assistance had been given in planning tours. In both cases the visitors were studying the health services in Australia.

Conditions of Service of Part-Time Medical Officers.

A letter was received from the New South Wales Branch drawing attention to an advertisement for part-time medical officers for an educational clinic at Canberra and asking that Federal Council take up with the Department of Health the conditions of service for such appointments. Federal Council resolved to take up the matter as requested.

WORLD MEDICAL ASSOCIATION.

Fourteenth General Assembly.

A copy of the minutes of the fourteenth General Assembly of the World Medical Association held in Berlin in September, 1960, was received from the Secretary-General. A report of the meeting was received from Dr. J. G. Hunter, the Federal Council delegate at the meeting, and a personal report from Dr. A. E. Lee. It was noted that Dr. L. R. Mallen of Adelaide had retired from the position of chairman of council and that Dr. J. G. Hunter had been elected as a member of the council.

Aid in Catastrophe.

A letter was received from the Secretary-General Elect forwarding a resolution adopted by the assembly directing its council to establish a programme for emergency care and requesting the closest cooperation of the national medical association in setting up a plan. The Federal Council expressed its support in principle of the proposals.

Training of Medical Personnel in Papua.

The General Secretary reported that strong criticism was expressed by delegates of the fourteenth General Assembly to certain aspects of the Commonwealth Government's plan to set up a medical school in Papua and to train indigenous medical practitioners. The matter had arisen out of the context of the regional report for Australia and Indonesia presented at the General Assembly by Dr. Hunter. The criticism was based on the grounds that the training of an inferior class of practitioner was an unwise and retrograde step. Dr. Hunter said that it was apparent that there was no realization of existing conditions in the Territory or of the outstanding success which a similar medical college in Suva, Fiji, had had over many decades. In a resolution on the subject the World Medical Association had recommended that the term "assistant medical officer" being used in connexion with those educated to work under the supervision of a medically qualified doctor be changed in accordance with the limited terms of reference they would be afforded and that this group be known as assistant social workers or technicians.

A letter was also received from Mr. R. W. Robson, Managing Director of Pacific Publications, Sydney, requesting the views of the Federal Council on the use of the title "doctor" for medical officers trained at the Suva Medical School.

After discussion the Federal Council expressed its support for the scheme for the training of medical personnel in Papua, but gave it as its opinion that the term "medical assistant" should be used instead of "assistant medical officer". It also resolved that the Department of Territories be urged to assist students in Papua and New Guinea to attain matriculation standard, so that early facilities (similar to those available to Colombo Plan students) might be provided for the training of such students in Australian medical schools to full graduate standard.

Report of Medical Ethics Committee.

A supplementary report of the Medical Ethics Committee of the World Medical Association dealing with human experimentation was received for comment. The Federal Council decided to approve the terms of the draft code of ethics on human experimentation subject to one amendment in wording suggested by the New South Wales Branch.

Documentary Film of Assembly.

The General Secretary reported that a copy was being purchased of a documentary film of the fourteenth General Assembly, and the Honorary Treasurer had arranged payment for this. The Federal Council approved the action of the Honorary Treasurer.

Fifteenth General Assembly.

The General Secretary reported that the fifteenth General Assembly of the World Medical Organization would be held in Rio de Janeiro, Brazil, from September 15 to 20, 1961. The Federal Council appointed Dr. L. R. Mallen and Dr. J. G. Hunter as delegates.

Hungarian Refugee Physicians Society.

The General Secretary referred to a memorandum from the World Medical Association on the "Hungarian Refugee Physicians Society". This matter had been brought to the attention of the Federal Council by the Victorian Branch, as a result of a request for assistance from the Society. The memorandum from the World Medical Association stated that there were only three or four Hungarian doctors presently in Austria who were not employed in financially gainful occupations, that these three or four doctors comprised the full membership of the "Hungarian Refugee Physicians Society", that this society was not registered with the Austrian Social Ministry or with the police, that the Social Ministry had been more than willing to cooperate with these doctors and had offered them any number of opportunities which they had not accepted, the Ministry reporting that they were very uncooperative, and that according to Austrian law these doctors had no legal rights to consider themselves as a society, or to solicit funds as they were not registered with the Social Ministry or the police.

W.M.A. Study Committee.

A copy was received of the report of the W.M.A. Study Committee which had been appointed by the General Assembly to consider the future activities of W.M.A. With the report were the comments of the Council of W.M.A. on the study committee's recommendations. The Federal Council received and approved the report.

Supporting Committee.

The Honorary Treasurer, Dr. W. F. Simmons, presented the financial statement of the Australian supporting com-

mittee of W.M.A. as at March 17, 1961. The credit balance as at August 3, 1960, had been £5344 8s. 2d. Contributions from the Branches since then had totalled £1484 13s. (£1244 3s. from the New South Wales Branch, and £240 10s. from the South Australian Branch), bringing the total to £6829 1s. 2d. Deduction of the expenses of the W.M.A. observer at the Sixth British Commonwealth Medical Conference and Annual Meeting of the British Medical Association in New Zealand left the total at £6677 14s. 11d.

Secretariat.

Dr. L. R. Mallen referred to the retirement of Dr. Louis Bauer as Secretary-General as from December 31, 1960, and advised of the unanimous decision of the council of W.M.A. to create a fund to show appreciation of the splendid work done by Dr. Bauer for W.M.A. The Federal Council resolved to contribute to the fund the sum of 20 U.S. dollars, the amount suggested by the council of W.M.A.

The Federal Council also decided to make a personal gift to Dr. Louis Bauer, the selection to be left in the hands of Dr. L. R. Mallen and the cost to be paid out of the funds of the supporting committee.

Reference was made to the appointment of Dr. Heinz Lord as Secretary-General of W.M.A. as from January 1, 1961, and to the sudden death of Dr. Lord on February 3, 1961.

Preparation of a Glossary.

A letter was received from the Secretary-General requesting a list of terms used locally which had defied easy translation or had given rise to misunderstanding after translation, with a request that precise definitions of those terms should be supplied. The letter had been referred to the Branches, and the matter was left in the hands of the General Secretary.

REPATRIATION DEPARTMENT.

Fees Payable to Private Radiologists.

In response to a request from the College of Radiologists of Australasia the Federal Council at its meeting in August, 1960, agreed to transmit to the Repatriation Department a request for an increase of 33⅓% in the schedule of fees payable to private radiologists. The General Secretary reported that the Repatriation Department had now advised that Treasury approval had been given for the increase in fees more or less as requested.

Sessional Fees Payable to Specialists.

At its meeting in August, 1960, the Federal Council had also endorsed a recommended increase in sessional fees payable to specialists rendering services on behalf of the Repatriation Department, the revised scale of fees having been drawn up at a meeting attended by representatives of the Federal Council, the Royal Australasian College of Surgeons, The Royal Australasian College of Physicians and the College of Radiologists of Australasia. The recommended scale had been sent to the Repatriation Commission, and the General Secretary reported that the Acting Chairman of the Repatriation Commission had now advised that the department had agreed to increase the sessional fees for specialists. However, the increased scale was less than that recommended by the Federal Council. The Colleges had been advised of the increased rates.

Radiological and Pathological Reports on Repatriation Patients.

In response to a recommendation of the Queensland Branch, the Federal Council at its meeting in September, 1959, decided to recommend to the Repatriation Commission that it should not be incumbent upon local medical officers to obtain permission before arranging radiological and pathological examinations for repatriation patients in areas where department facilities were unavailable. The General Secretary reported that the Commission had now agreed to this recommendation with certain provisos. The letter from the Acting Chairman of the Repatriation Commission dealing with this matter was published in the issue of March 25, 1961, at page 464.

Extension of Medical Services to Service Pensioners.

At its special meeting in November, 1960, the Federal Council expressed the opinion that: (i) the strongest exception should be taken to the Government's unilateral action in deciding to extend the scope of the present agreement to provide concessional services without prior discussions with the profession; (ii) the Minister's suggestion that the existing agreement be deemed to cover the additional class of patients until June 30, 1961, should be approved; but that (iii) it should be made perfectly clear to the Governmnt that this approval in no way committed the profession to any agreement which might be negotiated after June 30, 1961. The General Secretary reported that this matter had been passed on to the Minister for Repatriation, but so far no reply had been received.

Rates of Payment for Local Medical Officers.

At its special meeting in November, 1961, the Federal Council discussed the rates of payment to local medical officers and decided that the time was appropriate to commence negotiations for a revision of the existing agreement with the Repatriation Department. The matter was referred to the Branches, and after consideration of their views the Federal Council decided that a committee consisting of the President, Dr. H. C. Colville, and Dr. J. G. Johnson should wait upon the Chairman of the Repatriation Commission with reference to the establishment of a new agreement covering conditions of service of local medical officers. It also decided that the Committee should be requested to seek an increase in fees payable to local medical officers, fees for surgery consultation to be increased from 13s. to 15s. and fees for domiciliary visits from 15s. to 17s. 6d.

Prescription Forms.

A letter was received from the Secretary of the Repatriation Department setting out certain suggestions for the amendment of prescription forms and asking for comments. After consideration of the views of the Branches on the forms the Federal Council decided to approve the prescription form prepared by the Repatriation Department, with the exception of the size, which it recommended should be 8″ × 4½″ instead of 8″ × 5″, as the small form would fit more conveniently into the appropriate compartment in a widely used type of doctor's bag. It was also decided to ask the Repatriation Department to increase the number of repeats for prescriptions to two.

Pension Cards for Repatriation Patients.

A letter was received from the New South Wales Branch requesting Federal Council to ask the Repatriation Department to issue its pensioners with entitlement cards similar to those issued to war widows, listing their disabilities and thus giving the pensioners the right of free choice of doctors. After consideration of the views of the Branches, the Federal Council decided not to accept the recommendation of the New South Wales Branch.

Disclosure of Confidential Medical Information.

A letter was received from the New South Wales Branch requesting the Federal Council to take up with the Repatriation Department the matter of local medical officers being requested by the Department to supply confidential medical information on pensioners other than those with accepted disabilities. After discussion the Federal Council resolved that the question of the furnishing of such information without the consent of the patient be discussed with the Chairman of the Repatriation Commission.

DEPARTMENT OF SOCIAL SERVICES.

Sessional Fees Payable to General Practitioners.

At its meeting in September, 1959, the Federal Council decided to make an approach to the Department of Social Services requesting an increase in the sessional fees payable to general practitioners for rendering service on behalf of the Department of Social Services. The General Secretary reported that as yet no definite answer to this request had been received. However, in view of the fact that the Federal Council had subsequently decided to approach the Department of the Army for an increase in fees to a level in excess of that previously asked of the Department of Social Services, it was resolved to approach the latter department with a request for a scale of fees equal to that asked of the Department of the Army, namely £4 4s. for the first hour, £3 3s. for the second hour (£1 11s. 6d. for each half hour) and £2 2s. for each subsequent hour (£1 1s. for each half hour).

Form S.R.6.

At its meeting in August, 1960, the Federal Council decided to refer to the Director-General of Social Services certain amendments to the form S.R.6. The General Secretary reported that, according to advice received from the Principal Medical Officer of the Department, Dr. G. G. Burniston, a revision of the form was near completion.

Film: "Rehabilitation Adds Life to Years".

A letter was received from the Principal Medical Officer of the Department of Social Services advising of the availability for loan of the film entitled "Rehabilitation Adds Life to Years". This was described as a sound-colour film produced in 1959 by Reid J. Ray Film Industries, Minneapolis, for the American Medical Association's Committee on Rehabilitation and specifically for members of the practising medical profession. The film was designed to emphasize to the medical practitioner his all-important continuing responsibility in the complete rehabilitation of his patients and the value of using wherever possible existing rehabilitation services in the community for their comprehensive treatment, and indicated the type of disability requiring referral to a more elaborate rehabilitation centre. The film would be made available to any medical group wishing to borrow it for screening on application to the Senior Medical Officer of the Department of Social Services in the State concerned.

DEPARTMENT OF THE ARMY.

Medical Defence Cover for Members.

A letter was received from the South Australian Branch forwarding a request from the Directors of the Medical Defence Association of South Australia that the Crown be asked to define its policy in regard to medical defence cover for members. The request of the Directors of the Medical Defence Association was supported by the South Australian Branch. The Federal Council, after discussion, also decided to support the proposal.

ROYAL AUSTRALIAN AIR FORCE.

Appointment of Director-General of Medical Services.

The General Secretary reported that Air Commodore J. S. Wilson had been appointed to succeed Air Vice-Marshal E. Daley as Director-General of Medical Services of the Royal Australian Air Force.

MERVYN ARCHDALL MEDICAL MONOGRAPH FUND.

The Treasurer presented the financial statement of the Mervyn Archdall Medical Monograph Fund for the year ended December 31, 1960. This showed a credit balance of £2486. Since the previous statement £81 had been received in interest and £89 from the sale of Monographs Nos. 1 and 2.

DEPARTMENT OF LABOUR AND NATIONAL SERVICE.

Second National Conference on National Safety.

The General Secretary reported that Dr. C. J. Ross-Smith had attended the second National Conference on Industrial Safety in Canberra on February 21 and 22, 1961, as the representative of the Federal Council.

REHABILITATION.

A letter was received from Dr. D. Galbraith advising of correspondence with the Director-General of the Department of Social Services regarding the possibility of a further meeting of the Commonwealth Rehabilitation Advisory Committee and requesting Federal Council's view on the work of that committee and the provision of rehabilitation services in Australia. The Federal Council, after discussion, decided that Dr. Galbraith should be asked to interview the Director-General of Social Services for the purpose of discussing *inter alia* the terms of the Commonwealth Rehabilitation Advisory Committee.

SHIPS' SURGEONS.

A letter was received from the New South Wales Branch recommending that the matter of an increased remuneration for ships' surgeons be taken up again with the Australasian Steamship Owners' Federation. The Federal Council decided to accede to the request of the New South Wales Branch and also to take up the matter of the medical equipment provided by shipping companies.

DEATH OF GOVERNOR-GENERAL.

The General Secretary reported that on behalf of the Parent Body and the Federal Council a message of sympathy had been sent to Her Excellency the Viscountess Dunrossil, at the time of the death of the Governor-General of the Commonwealth of Australia, His Excellency the Viscount Dunrossil of Vallaquie.

VOTES OF THANKS.

The thanks of the Federal Council were extended to the Council of the New South Wales Branch for its hospitality and its offices and to Dr. and Mrs. R. H. Macdonald for their hospitality. Thanks were also extended to the New South Wales representatives for their hospitality and to Dr. J. G. Hunter, Dr. C. J. Ross-Smith and Miss H. Cameron for their services during the meeting. The thanks of the meeting were extended to the President, Dr. H. C. Colville, for presiding.

DATE AND PLACE OF NEXT MEETING.

It was decided that the date and place of the next meeting be left in the hands of the President.

Out of the Past.

A PROPOSED SCHOOL OF TROPICAL MEDICINE.

[From the *Australasian Medical Gazette*, March 20, 1902.][1]

From the Proceedings of the Section of Medicine at the Intercolonial Medical Congress Sixth Session, Hobart, 1902.

DR. GOLDSMITH [Northern Territory] then submitted the following report of the committee consisting of Drs. Ham (Brisbane), Blackburn (Sydney), and Goldsmith appointed at a former meeting of the Section, on a School of Tropical Medicine:

"The Committee consider it desirable that a School of Tropical Medicine and Research should be established for the scientific and systematic investigation of tropical diseases in Australia, and to this end they recommend the following:

"1. That Brisbane would be the most suitable place to carry out such investigations.

"2. That the proposed school should be established on lines similar to those of the School for Tropical Medicine in London.

"3. That the Rt. Hon. the Premier of the Commonwealth, and the Premiers of the various States, should be officially approached by the Executive of the Congress for grants to enable such an institution to proceed with its work, and that the Rt. Hon. the Secretary of State for the Colonies, and Dr. Patrick Manson, the head of the School of Tropical Medicine in London, be communicated with on the subject.

"4. The section recommends the Congress to appoint Dr. Goldsmith as its representative in England."

The report was adopted and ordered to be sent on to the Congress.

Correspondence.

PERSONALITY AND TRAFFIC ACCIDENTS.

SIR: Today there are more cars and other motor vehicles and the speed of all these vehicles is greater than formerly. There are in consequence more traffic accidents, both to drivers and pedestrians. Usually, in discussing traffic accidents, the personality characteristics and mental and physical states of the persons involved in these accidents, tend to be disregarded.

The driver may have an elated emotional state of divers origin. He may show signs of mental immaturity. He may have the instinct of rivalry in a pathological degree. The effects of alcohol are well known; alcohol may cause intoxication, delusional states, mental deterioration, Korsakoff's disease and damage to the higher faculties of the brain. Common results of alcoholism are recklessness, lack of judgement and delayed reaction time.

Various diseases may affect driving ability, among them diabetes with symptoms resulting from acetonæmia or from hypoglycæmia. The latter condition may develop rapidly. Various drugs are known to have a deleterious effect on driving. Epilepsy can be a bar to safe driving. Nocturnal epilepsy and the vasovagal epilepsy of Gowers may render the driver a menace on the road. Colour blindness can be a danger, and it must be remembered that 1% of males are markedly red-green colour-blind. Routine tests used by the police to decide if a person is under the influence

[1] From the original in the Mitchell Library, Sydney.

of alcohol can be inadequate as tests of judgement and of ability to drive a car.

Ignorance can be a cause of traffic accidents. High speeds around curves may indicate a lack of knowledge of one of Newton's laws—the lack of knowledge that high speeds cause heating of tyres, which heat may cause softening of nylon cords used in some tyres in preference to cotton and blow-outs may become almost inevitable under such circumstances.

Pedestrians may be involved in accidents because of alcohol, poor vision, ill-health, senility and other causes.

Apart from the various mental and physical states, the main cause of traffic accidents is speed, with consequential inability to act quickly, or to stop within a short distance.

In railroad practice it is accepted that no buffers can protect a train travelling at more than 30 miles per hour. What measure of safety has the car?

Yours, etc.,

Melbourne.
March 29, 1961.

S. J. Cantor.

AN APPEAL FOR JOURNALS.

Sir: The problem of adequately equipping the journal section of a library of a new hospital is most acute in the first years.

The Queen Elizabeth Hospital in Adelaide was opened for patients in 1959. It is the second University teaching hospital in the city, and has a total of 500 beds. There is a fine new library, but it has been difficult to acquire bound volumes of back numbers of journals for the shelves. It is thought that there may be doctors or others among your readers who would like to donate bound volumes of journals covering all or part of the last 20 years.

The hospital would be most grateful if prospective donors would write with details to the Medical Superintendent. Any transport costs for acceptable volumes would be borne by the hospital.

Yours, etc.,

The Queen Elizabeth Hospital,
Woodville,
South Australia.
March 27, 1961.

R. Douglas Carman,
Medical Superintendent.

PULMONARY HYDATID DISEASE.

Sir: The article "Pulmonary Hydatid Disease" appearing in your Journal of March 11, 1961, serves to emphasize the continued presence of a disease which in a large measure is preventable. It is desired, however, to comment on that paragraph of the article relating to prevention, which states *inter alia* that the infestation of offal-fed dogs can be avoided by ensuring that the offal is boiled. It is the writer's impression that in practice slaughterhouse offal would seldom be boiled before being fed to dogs, and that when this precaution is taken the period of boiling is relatively short. Fastier[1] has shown that, when a sheep's liver is placed in water already boiling, 40 minutes' immersion was required before the scolices could be assumed to be destroyed, and if placed in cold water and brought to the boil sterilization could not be considered complete until 30 minutes after boiling commenced. It is submitted that sheep's offal is seldom, if ever, exposed to boiling temperatures for these periods of time, and serious doubt, therefore, exists as to the desirability of a recommendation to stockowners that offal is made safe if boiled.

In the light of this information, the writer is of the opinion that the use of adult sheep's offal as dog food, even when boiled to the satisfaction of the landholder, is not a justifiable risk. The value of the offal is so small and the incidence of hydatids so high that the use of alternative foods is well justified until the present incidence of hydatids is markedly reduced.

Yours, etc.,

Department of Agriculture,
Hobart,
Tasmania.
March 30, 1961.

G. K. Meldrum,
Chief Veterinary Officer.

[1] *Parasitology*, 1949, 39: 157.

Obituary.

JOHN GERALD HAYDEN.

We are indebted to Dr. H. G. Furnell for the following account of the career of the late Professor J. G. Hayden.

John Gerald Hayden was born in Ballarat in 1901, the eldest of six children. This charming old mining city has given many distinguished men to Australia; their contribution to our welfare is probably far greater than the value of the gold produced in Ballarat's Golden Age. It is interesting that while John was President of The Royal Australasian College of Physicians, Douglas Miller, also born in Ballarat, was President of the Royal Australasian College of Surgeons; they had attended the same school as very small boys. Another famous native of Ballarat with whom I had the good fortune to be closely associated was Sir Leslie Morshead, and strangely enough the Hayden and Morshead families had some business associations in Ballarat.

John was a true son of Ballarat, as his father and his mother were born there, the parents of both having gone to Ballarat in the 1850's. His maternal grandmother was English, the other grandparents were Irish. When John was eleven years of age his father died, and his mother, without brothers or sisters to help, brought up and educated her six children—all to fill positions of importance in the community. One brother is a priest, one a successful business man in Ballarat, the youngest, Frank, is a leading gynæcologist in Melbourne; of his sisters one is married, and the younger is the Mother Superior of Mary's Mount Convent in Ballarat. This is truly a proud record for a young widow. In spite of her early difficulties Mrs. Hayden lived to a venerable old age, and was able to enjoy life thoroughly to its end and to see with pride the highly satisfactory results of her devotion.

John's school was St. Patrick's College, Ballarat, of which he was *dux*; there he won a scholarship to Newman College within the University of Melbourne. After a distinguished undergraduate career, he became a resident medical officer at St. Vincent's Hospital, thus commencing a long life of service to that institution, which places him very high in the list of distinguished men who have made St. Vincent's Hospital Clinical School what it is today. His early post-graduate career was that of many young men destined for success: resident, registrar (in this year he qualified for the degree of M.D. Melbourne, with obstetrics his special subject); then to America, where he worked at St. Louis with Evarts Graham, and to England, where he gained the M.R.C.P. diploma; and finally back to Australia in 1928, where, at the age of 27 years, he started practice as a consulting physician.

He took rooms with Sir Hugh Devine, and his brother tells me that he earned his living mostly after 6 p.m., when Sir Hugh would go home, leaving the remaining patients to John—those with medical troubles to be retained, and the surgical patients to be referred back to his principal.

John made two subsequent visits to Europe and the United States of America, in 1939 and 1954. His professional career was a story of unbroken success. He was appointed out-patient physician in 1928 and in-patient physician in 1934; he became a Fellow of the Royal College of Physicians in 1941, and was a Foundation Fellow of The Royal Australasian College of Physicians. The highlights of his career were the presidency of The Royal Australasian College of Physicians in 1958, and the invitation to the chair of medicine at St. Vincent's Hospital Clinical School in the University of Melbourne.

This truly is a dazzling record; but the man was far greater than these achievements. As he grew older, he grew wiser—not a universal habit—and his value to the community was steadily increasing; his untimely death has deprived us of an erudite physician, a great teacher and a very wise counsellor. Throughout his life he gave service fully and gladly to any cause with which he was associated. I do not propose to give any details of his hospital and University work, which will be covered elsewhere. In the post-war period, particularly while he was Stewart Lecturer in Medicine, I used to meet him on many committees, but only a fraction of those on which he served, and I wondered how he possibly had time to earn a living.

Early in his career he realized that war was probable at some future date, and he devoted part of his scanty leisure to the Australian Army Medical Corps, with the result that when war broke out he was a well-trained field ambulance

commander. However, his ability as a physician was regarded as the most valuable asset to the service, and he went away as O.C. Medical Division 2/7 Australian General Hospital; later, in Egypt, he succeeded Colonel W. Summons as Commander of the Unit, with which he served till the end of the war. His unit gave signal service in Egypt, where it received all our casualties from Alamein, and later at Lae in New Guinea. For his service in the Middle East he was created a Companion of the Most Excellent Order of the British Empire. His army associations continued after the war, and for many years prior to his death he had been consulting physician to the army and to the Repatriation Department.

While his professional career was one of unbroken success, his private life held many and grave set-backs. His first wife tragically disappeared in 1947, having been for some time in poor health. With the courage he so amply demonstrated later, he bore this, and continued with his work, both public and private, in a manner that earned the admiration of his friends. He and his wife had adopted a daughter, who was a great consolation to him at this time. In 1952 he married Mrs. Patricia Bell, and is survived by his widow and infant son. This latter period was the happiest of his life, and everything appeared to combine to give him the security and content he so thoroughly deserved, when his fatal illness struck.

John's health of later years had not been good. He had had great trouble with a penetrating ulcer, which was treated by gastrectomy in 1955 with a splendid result. Before this operation a very serious possibility of malignant disease had to be discussed with his surgeon. His courage in this crisis gave a foretaste of the fortitude he displayed in such ample measure in his final illness.

John Hayden was a thoroughly pleasant companion, with a good sense of humour and a highly intelligent interest in affairs in general. He was a fairly wide reader, quick and retentive, but his work was his real hobby. He played golf, not enthusiastically, and of late had become interested in trout fishing, a contemplative pursuit which seems to be particularly favoured by physicians. One subject that interested him considerably was painting, and he was always ready to talk with knowledge, particularly of Australian artists. The theatre also gave him pleasure, and he was a regular patron of this form of entertainment. He was excellent as both host and guest, and thoroughly enjoyed social activities.

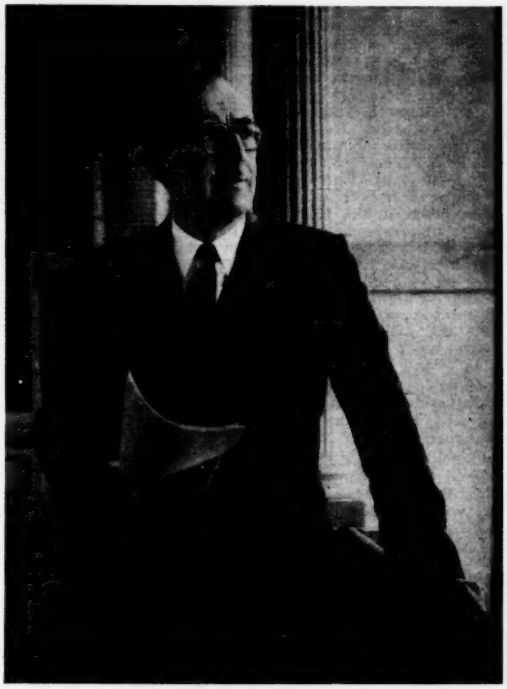
His real interest was in people, particularly young people; he truly liked teaching, and fortunately was a master of this difficult art. His clinics and lectures, post-graduate and undergraduate, were always most popular with students; this is only one direction in which his loss will be deeply felt. His concern with the young was deep and persistent; any young man of promise was assured of very practical and lasting help from him. Many of the young leaders of the profession today owe a great deal to his encouragement, advice and assistance in finding suitable hospital posts. He leaves behind him an imposing array of young men deeply conscious of the debt they owe him, and sincerely grateful for his wise help in important stages of their careers. I asked a young physician, of late very closely associated with John, what he thought were his outstanding qualities. Without hesitation the answer was, first, his outstanding integrity, and next his amazing fortitude in his final illness. This I think is a very sound appreciation of a brave and gifted man. John Hayden could truly have said of himself

> I warmed both hands before the fire of life;
> It sinks, and I am ready to depart.

To his widow and family goes the deep sympathy of the profession which he served so truly, and which held him in high and affectionate regard.

MR. ANTHONY R. KELLY, Dean of the Clinical School, St. Vincent's Hospital, Melbourne, writes: With the death of Professor John Gerald Hayden on December 26, 1960, St. Vincent's Hospital in Melbourne lost one of her most brilliant sons, and the graduate whose work for the hospital, as well as for several other important organizations, has been outstanding and unsurpassed. So, at this time, all connected with St. Vincent's experience a great sense of grief at the loss of such a good friend.

After being *dux* of the school at St. Patrick's College, Ballarat, and completing the pre-clinical years of the course, John Hayden first came to St. Vincent's as a student in 1921, and from that time, except for periods of study abroad and service in the army in World War II, was intimately associated with the hospital until his death, which took place in his own Department of Medicine within the hospital. As an undergraduate, his work was of a very high order, and he graduated with honours in 1923. Until 1925 he was a member of the resident medical staff, and then, after a period of post-graduate study, during which he obtained the M.D. (Melbourne) in 1925 and M.R.C.P. (London) in 1927 (elected F.R.C.P. in 1941), he was appointed to the honorary staff as a physician to out-patients in 1928. Six years later, in 1934, he was appointed physician to in-patients, a position he occupied until 1956, when he was invited by the University of Melbourne to fill the chair, and establish the University of Melbourne Department of Medicine at St. Vincent's Hospital. This he did with characteristic efficiency before his final illness and death.



John Hayden will long be remembered at St. Vincent's Hospital for many outstanding qualities; but it is to the great physician and teacher of medicine and professional colleague that I pay particular tribute on this occasion. He was undoubtedly an outstanding physician. His knowledge of medicine when he was first appointed to the staff in 1928 was great, and this he enlarged and kept up to date, and modified in the light of his enormous experience with the passage of the years; so that one could solicit his opinion on any aspect of medicine, or consult him on any difficult clinical problem, confident that the opinion he gave would be in accord with the very best thought and practice in the subject. In a large public hospital and private practice over a period of 30 years one cannot assess the number of patients and their relatives and their medical advisers who have been, and are, indebted to John Hayden's medical competence and skill; but the large and widely representative crowd present at his requiem and funeral on an extremely hot day in the middle of the holiday period bears witness that it is very great indeed.

However, despite his great eminence as a consultant, it is probably as a teacher of medicine that John Hayden will be best remembered by the greatest number of his fellow medical graduates. For over 30 years he was a great teacher of students at St. Vincent's Hospital, and every graduate of the hospital during that period (between six and seven hundred) acknowledges the great debt owed to him. But it was not only in undergraduate teaching that he rendered such outstanding service to medicine, but also in the teaching of his speciality to post-graduate students. Year in and year out his ward rounds have been attended by those seeking the knowledge necessary to acquire higher degrees and diplomas in medicine. Over the years these groups have sometimes been small, sometimes large, and have consisted not only of graduates of the St. Vincent's Hospital Clinical School, but also of those from the other

clinical schools in this city, from other States and from other countries. All these men and women knew that from John Hayden they would learn medicine at the highest level, and he did not disappoint them. In this age, when so much consideration is being given, opinions are being expressed and advice is being offered on the technique of teaching, including clinical teaching, it is interesting to analyse the technique of this outstanding teacher. This is very easy indeed; it consisted in a thorough knowledge of his subject and the ability to expound it clearly and authoritatively, with a leavening of the Socratic method. His manner, though not strict, was such that students tended to listen attentively rather than not; then, if they had the necessary intelligence and wanted to learn, they did so.

As well as in the practice and teaching of medicine, John Hayden served St. Vincent's Hospital in many roles over the years. He was at various times Sub-Dean and Dean of the Clinical School. He was a member of the Hospital Advisory Council, and of the executive of the John Holt School of Medical Research, and represented the hospital on innumerable committees, sub-committees and deputations. In all these roles the brilliance of his intellect, combined with that human understanding, friendliness and tact which were characteristic of him, resulted not only in the views of the hospital being suitably presented and properly understood, but also in its prestige being raised at the same time.* This understanding and friendliness were characteristic of John Hayden on all occasions, making him a most popular colleague, and these qualities were particularly helpful to his younger associates, who always found him approachable, and to whom he was ever ready to give the great benefit of his advice, opinion, knowledge and experience.

Besides his work at St. Vincent's, John Hayden also performed particularly outstanding service for the army, the University of Melbourne and The Royal Australasian College of Physicians. Others will undoubtedly write on these aspects of his life; but, at this time, I would like to acknowledge the honour he incidentally brought to St. Vincent's Hospital in all these activities.

John Hayden's death leaves a hiatus at St. Vincent's which will be extremely difficult and, at the present time, seems impossible, to fill. On behalf of the hospital I pay our public tribute and express our gratitude for everything he has done for us in the past 40 years; and to his family, particularly to his wife, Patricia, and to his brother, our gynæcologist colleague, Mr. Frank Hayden, we tender our most sincere sympathy.

Sir Albert Coates writes: Professor John G. Hayden, C.B.E., was one of the oustanding products of the Medical School of the University of Melbourne in the early 1920's—a brilliant student, who early showed great promise, later to be fulfilled in the teaching and administrative fields. He had not reached the peak of success to which he was destined. He was cut off in full flower, at a time when his academic eminence and his great gifts as a medical statesman were making their mark in Australia.

As he was a colleague whom I had known and admired since early association as a junior officer of the Australian Army Medical Corps in 1926, it was for me an honour and a pleasure to be appointed as his running mate in the office of Stewart Lecturer, he in medicine, I in surgery, in the University of Melbourne in 1942. For seven years we had an academic association befitting the heads of departments in such an institution. This twilight period before the dawn of professorial clinical appointments in our medical school was marked by much discussion and frequent debate.

John Hayden acquitted himself with honours in those days. A noble character, with a breadth of vision not always found in one so placed, he was a pillar of strength and a fount of wisdom. His *savoir-faire*, broad common-sense and innate modesty contributed in no small measure to the solution of the knotty problems peculiar to the Melbourne Medical School at that time. He played an historic part in the establishment of the clinical chairs; but only after the two first chairs were filled (James Stewart Professorships in Medicine and Surgery) would he consent to offer himself as candidate for the second chair of medicine. To the delight of friends and professional colleagues, he was appointed Professor of Medicine, with the department located in his own beloved St. Vincent's Hospital. That magnificent institution in its modern form owes much to his foresight and skilful planning. Others will record his great work there.

As one who sat with him on many committees—University, Repatriation Department and advisory boards—I had the feeling of being in the presence of a superior man. We both had our hospital loyalties, but the overall good was paramount in Hayden's mind and his ultimate decision.

As a host, adorning his office, whether professor, president of The Royal Australasian College of Physicians or semi-public medical adviser, he was typical of the educated gentleman. His wife, Pat, gave a dignity and charm to the parties hospitably arranged for overseas and interstate guests. The last one was notable for the courage and composure of both those heroic people. In the full knowledge of the approaching separation, they kept the flag flying. John continued his work in the final examinations in medicine. Three weeks later he laid down his burden. A scholar, a learned doctor, a beloved physician—he has passed from amongst us.

Many of us feel a deep sense of personal loss, but are sensible of having had our lives enriched by our association and friendship with the late Professor John Hayden.

Dr. G. C. de Gruchy writes: It has been my privilege since I first met Professor Hayden as a resident medical officer in 1945 to be closely associated with him in clinical and teaching matters, particularly during the past two years when I worked with him in the Department of Medicine at St. Vincent's Hospital. Professor Hayden was an outstanding clinician and teacher, who could inspire a real enthusiasm for medicine in young post-graduates. As a clinician he was at home in all branches of internal medicine, and he was able to talk with medical specialists about their specialty on close to equal terms. As a teacher he was clear, practical and authoritative. One of his particular teaching skills was his ability to make an undergraduate round stimulating for post-graduates without interfering with the basic instruction of the undergraduates; for this reason his clinic was always attended by post-graduates, particularly in the years 1945 to 1950, when the very large retinue following him was one of the features of the medical wards at St. Vincent's. Professor Hayden's interest in post-graduates did not stop with teaching; he was always ready to advise and assist properly trained young physicians in the planning of their careers and in obtaining hospital appointments. Many of Melbourne's younger physicians from all clinical schools acknowledge with gratitude the help which he gave them.

Professor Hayden was a first-class administrator. His absolute integrity, sound judgement and strong sense of duty were widely known, and for this reason he was sought for numerous administrative tasks in many fields. When a decision had to be made, he carefully assessed the evidence and gave a prompt decision based on the merits of the case. He always gave a sympathetic hearing to any reasonable proposition, and if he decided that it deserved support, did his best to help. However, he could not be influenced by vague or second-rate propositions. Part of his success as an administrator was that he liked administration.

The first reaction of people who worked with him was of respect and admiration; but on closer association, to these was added affection. His fine qualities brought forth a very warm response and a feeling of personal loyalty in his staff, both medical and non-medical, which resulted in a strong team spirit in his department.

Professor Hayden's fortitude during his last illness was remarkable. Once the diagnosis of his disorder was established, his policy was to work until the end; this he did, despite great pain and physical disability. During the last months of his life he worked constantly on committee matters relating to medical education, and for the betterment of his hospital and the assistance of young members of the profession. Professor Hayden's memory will long be honoured by his students and colleagues.

Dr. J. J. Billings writes: It is one of the privileges of our profession to attend a saintly man or woman at the hour of death, and John Hayden was such a man. When he fisrt entered medical practice, his intellectual eminence was quickly recognized, not only at St. Vincent's Hospital, where his colleagues were proud of him, but in his energetic work within the University of Melbourne and The Royal Australasian College of Physicians. His achievements were so great that it was possible to overlook the simple integrity of his character; yet his memory is held in warm affection as well as gratitude.

It was in his final illness that his courage and spiritual serenity revealed themselves and gave him a marvellous dignity. These virtues were fostered by his deep religious faith, which he practised constantly, without ostentation, and which moulded his character throughout his life. His true genius lay in this admirable balance of all his faculties.

The College of Radiologists of Australasia.

EXAMINATION RESULTS.

THE College of Radiologists of Australasia announces that the following candidates were successful in the March, 1961, examinations, Part II, for membership of the College:

Radiodiagnosis: Dr. W. Lowen (Victoria), Dr. J. P. Masel (Queensland), Dr. D. E. Nairn (South Australia), Dr. R. J. Thorpe (Queensland).

Radiotherapy: Dr. R. A. Hurley (Victoria), Dr. H. R. Withers (Queensland).

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

COURSES DURING APRIL AND MAY, 1961.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that the following courses will be conducted during April and May, 1961.

Week-End Course in Rheumatic Diseases.

A week-end course in rheumatic diseases will be held under the supervision of Dr. Selwyn Nelson on Saturday and Sunday, April 22 and 23, 1961, in the Scot Skirving Lecture Theatre of the Royal Prince Alfred Hospital. The guest lecturer will be Professor E. G. L. Bywaters, F.R.C.P., Professor of Rheumatology, Postgraduate Medical School, University of London, and Honorary Consultant Physician, Canadian Red Cross Memorial Hospital, Taplow. The programme will be as follows:

Saturday, April 22, chairman, Dr. Selwyn Nelson: 2 p.m., Opening remarks by Chairman; 2.5 p.m., symposium, "Treatment of Arthritis (Excluding Steroids)": "The Scope of the Problem", Dr. Selwyn Nelson; "Drugs in the Treatment of Arthritis", Dr. John Sands; "Surgical and Orthopædic Treatment", Dr. N. H. Morgan; "X-Ray Therapy", Dr. H. J. Ham; "Physical Methods of Treatment", Miss P. M. Lee (physiotherapist); quiz session.

Sunday, April 23, chairman, Dr. Ralph Reader: 9.30 a.m., case presentations, illustrating various problems in diagnosis and management, Dr. F. Harding Burns and Dr. Ralph Reader; 10.45 a.m., symposium, "Steroids"—Dr. R. G. Robinson, Dr. John Sands, Dr. John Greenaway; 11.45 a.m., "Vascular Lesions in Connective Tissue Disease", Professor E. G. L. Bywaters.

Sunday, April 23, chairman, Dr. Selwyn Nelson: 2 p.m., "Joint Lesions in Bone Disease", Professor E. G. L. Bywaters; 3.30 to 5 p.m., symposium, "Rheumatoid Arthritis" (introduced by *The Voice of Medicine*, a recorded panel discussion in The Hague, Holland—Dr. R. Lamont Havers *et alii*), Professor E. G. L. Bywaters, Dr. P. J. Benjamin, Dr. Brian Haynes, Dr. R. G. Robinson, Dr. Naomi Wing.

The fee for attendance is £3 3s., and early written application should be addressed to the Course Secretary, The Post-Graduate Committee in Medicine, Herford House, 188 Oxford Street, Paddington, N.S.W. Telephone: FA 0671. Telegraphic Address: "Postgrad Sydney".

Annual Subscription Course: Anæsthetic Demonstrations and Tutorials.

Professor T. Cecil Gray, F.F.A.R.C.S., D.A., Professor of Anæsthetics in the University of Liverpool, and Sims Commonwealth Travelling Professor for 1961, will conduct practical demonstrations and tutorials in anæsthesia at Sydney Hospital on May 5, 8, 9 and 10, 1961. At 8.30 each morning Professor Gray will conduct a practical demonstration, and at 2 o'clock each afternoon he will hold informal tutorials. The morning demonstrations, which will be held in the Maitland Operating Theatres of the Hospital, will be strictly limited to specialist anæsthetists. Those wishing to attend are requested to advise the Committee not later than May 2, 1961. Attendance at the afternoon tutorials, which will be held in the Maitland Lecture Hall, is unlimited, and all medical practitioners are invited to attend.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED MARCH 11, 1961.

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	..	..	..	..	..	..	..	..
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	4	..	5(5)	..	..	..	7	..	16
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	1(1)	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	3(2)	10(9)	5(5)	..	..	1	10	..	29
Diphtheria	..	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	..	..	1(1)	4(4)	2(1)	1(1)	..	..	8
Encephalitis	1(1)	1	..	..	..	..	..	..	2
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	116(44)	53(33)	14(7)	28(8)	10(4)	6(1)	2	2	231
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	3	..	..	..	..	..	3
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	..	1	..	..	..	..	..	..	1
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	1	1(1)	..	..	..	..	2
Paratyphoid	..	1(1)	..	..	..	..	..	..	1
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	1	5(1)	..	..	..	..	..	..	6
Puerperal Fever	2	..	..	..	..	..	..	..	2
Rubella	..	5(3)	..	1(1)	9(6)	..	..	..	15
Salmonella Infection	..	..	..	..	2(2)	..	..	..	2
Scarlet Fever	3(3)	9(7)	5(5)	1(1)	..	4	..	..	22
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	4	..	..	..	4
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	24(16)	12(11)	5(3)	5(5)	..	3(1)	..	..	49
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Professor Gray will also give a lecture in the annual subscription course entitled "Accident and Design in Surgery", on Thursday, May 18, at 8.15 p.m., in the Stawell Hall, 145 Macquarie Street, Sydney.

This programme is being held by arrangement with the Faculty of Anæsthetists, Royal Australasian College of Surgeons (N.S.W. State Committee). Inquiries should be made to the Post-Graduate Committee in Medicine, at the address given above.

SCHOOL OF PUBLIC HEALTH AND TROPICAL MEDICINE AND THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Post-Graduate Course in Occupational Health.

A FULL-TIME COURSE in occupational health will be held at the School of Public Health and Tropical Medicine, University of Sydney, from July 24 to August 11, 1961. The course, which is primarily designed for medical graduates taking the Diploma in Public Health, will comprise a series of lectures and visits to factories and other industrial undertakings. The main subjects will be as follows: definition and scope of occupational health; historical aspects; activities in other countries, and of international organizations; developments in Australia; organization, functions and responsibilities of occupational health services; duties and responsibilities of industrial medical officers; the working environment—chemical and physical aspects; recognition of occupational factors in disease and disability; psychological problems in industry; diseases and other effects caused by dusts, metals, solvents, pesticides and other chemicals; occupational diseases of the skin; industrial ophthalmology; noise and occupational hearing loss; ionizing radiations—occupational and public health aspects; the prevention of occupational disease; accident causation and prevention; rehabilitation and employment of disabled persons; occupational health legislation; introduction to laboratory equipment and procedures. Visits will be made to representative industries in order to study working conditions and processes related to the subjects of the course. The visits will occupy approximately twelve half-day periods.

Any medical practitioner interested in occupational health may apply to attend either the complete course, or the lectures only. No fees will be charged. Written applications should be sent to the Director, School of Public Health and Tropical Medicine, University of Sydney. The number of applicants for full-time attendance will be limited to 12; and the closing date for receipt of these applications is June 30, 1961. Applications to attend lectures only may be received later than that date. Further particulars may be obtained from Dr. Gordon C. Smith, supervisor of the course, at the School of Public Health and Tropical Medicine.

Notes and News.

Trends in Medicine-Church Relations.

The Clergy-Doctor Commission of the Australian Council of Churches is sponsoring a seminar entitled "Trends in Medicine-Church Relations", to be held at the University of New South Wales on Saturday, June 3, 1961, from 9.30 a.m. to 5.30 p.m. The speakers will be Dr. Basil Hetzel, Reader in Medicine at the University of Adelaide, and the Reverend John Garrett, Warden of the Congregational Theological College, Sydney. There will be group and plenary discussions, and lunch and refreshments will be provided. Doctors and clergy of all faiths are invited. Application for registration should be made to the Reverend J. Brand, 136 Edenholme Road, Abbotsford, by May 24, and should be accompanied by the seminar fee of £1 1s.

Appointment as Consultant to Lederle Laboratories.

Dr. A. J. Metcalfe has been appointed Consultant to Lederle Laboratories Products, suppliers of ethical drugs and pharmaceuticals.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Long, Geoffrey Joseph, M.B., B.S., 1959 (Univ. Sydney), St. Vincent's Hospital, Darlinghurst.

Chang, Peter Nang-Sang, M.B., B.S., 1960 (Univ. Sydney), 83 Griffith Avenue, Bankstown.

Diary for the Month.

APRIL 15.—Queensland Branch, B.M.A.: Seventh Branch Convocation.
APRIL 17.—Victorian Branch, B.M.A.: Finance Subcommittee.
APRIL 18.—New South Wales Branch, B.M.A.: Medical Politics Committee.
APRIL 19.—Western Australian Branch, B.M.A.: General Meeting.
APRIL 19.—Victorian Branch, B.M.A.: Clinical Meeting.
APRIL 20.—New South Wales Branch, B.M.A.: Clinical meeting.
APRIL 20.—Victorian Branch, B.M.A.: Executive of Branch Council.
APRIL 21.—New South Wales Branch, B.M.A.: Ethics Committee.
APRIL 26.—Victorian Branch, B.M.A.: Branch Council.
APRIL 26.—New South Wales Branch, B.M.A.: Hospitals Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

Authors of papers are asked to state for inclusion in the title their principal qualifications as well as their relevant appointment and/or the unit, hospital or department from which the paper comes.

References to articles and books should be carefully checked. In a reference to an article in a journal the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of article. In a reference to a book the following information should be given: surname of author, initials of author, year of publication, full title of book, publisher, place of publication, page number (where relevant). The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full data in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this Journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in Australia can become subscribers to the Journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £6 *per annum* within Australia and the British Commonwealth of Nations, and £7 10s. *per annum* within America and foreign countries, payable in advance.